Control of Emissions from Marine SI and Small SI Engines, Vessels, and Equipment

Final Regulatory Impact Analysis

Chapter 2
Air Quality, Health, and Welfare Concerns

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CHAPTER 2: Air Quality, Health, and Welfare Concerns

The standards finalized in this action will reduce emissions of hydrocarbons (HC), oxides of nitrogen (NOx), carbon monoxide (CO) and air toxics from the engines, vessels and equipment subject to this rule. Emissions of these pollutants contribute to ozone, PM and CO nonattainment and to adverse health effects associated with air toxics. The emissions from these engines, vessels and equipment also contribute to adverse environmental effects.

The health and environmental effects associated with emissions from Small SI engines and equipment and Marine SI engines and vessels are a classic example of a negative externality (an activity that imposes uncompensated costs on others). With a negative externality, an activity's social cost (the cost on society imposed as a result of the activity taking place) exceeds its private cost (the cost to those directly engaged in the activity). In this case, as described in this chapter, emissions from Small SI engines and equipment and Marine SI engines and vessels impose public health and environmental costs on society. The market system itself cannot correct this externality. The end users of the equipment and vessels are often unaware of the environmental impacts of their use for lawn care or recreation. Because of this, consumers fail to send the market a signal to provide cleaner equipment and vessels. In addition, producers of these engines, equipment, and vessels are rewarded for emphasizing other aspects of these products (e.g., total power). To correct this market failure and reduce the negative externality, it is necessary to give producers social cost signals. The standards EPA is finalizing will accomplish this by mandating that Small SI engines and equipment and Marine SI engines and vessels reduce their emissions to a technologically feasible limit. In other words, with this rule the costs of the services provided by these engines and equipment will account for social costs more fully.

In this Chapter we will discuss the impacts of the pollutants emitted by Small SI engines and equipment and Marine SI engines and vessels on health and welfare, National Ambient Air Quality Standard (NAAQS) attainment, and personal exposure. Air quality modeling and monitoring data presented in this chapter indicate that a large number of people live in counties that are designated as nonattainment for either or both of the 8-hour ozone or $PM_{2.5}$ NAAQS. Figure 2-1 illustrates the widespread nature of the ozone and $PM_{2.5}$ nonattainment areas and also depicts mandatory class I areas. The emission standards in this rule will help reduce HC, NOx, PM, air toxic and CO emissions and their associated health and environmental effects.

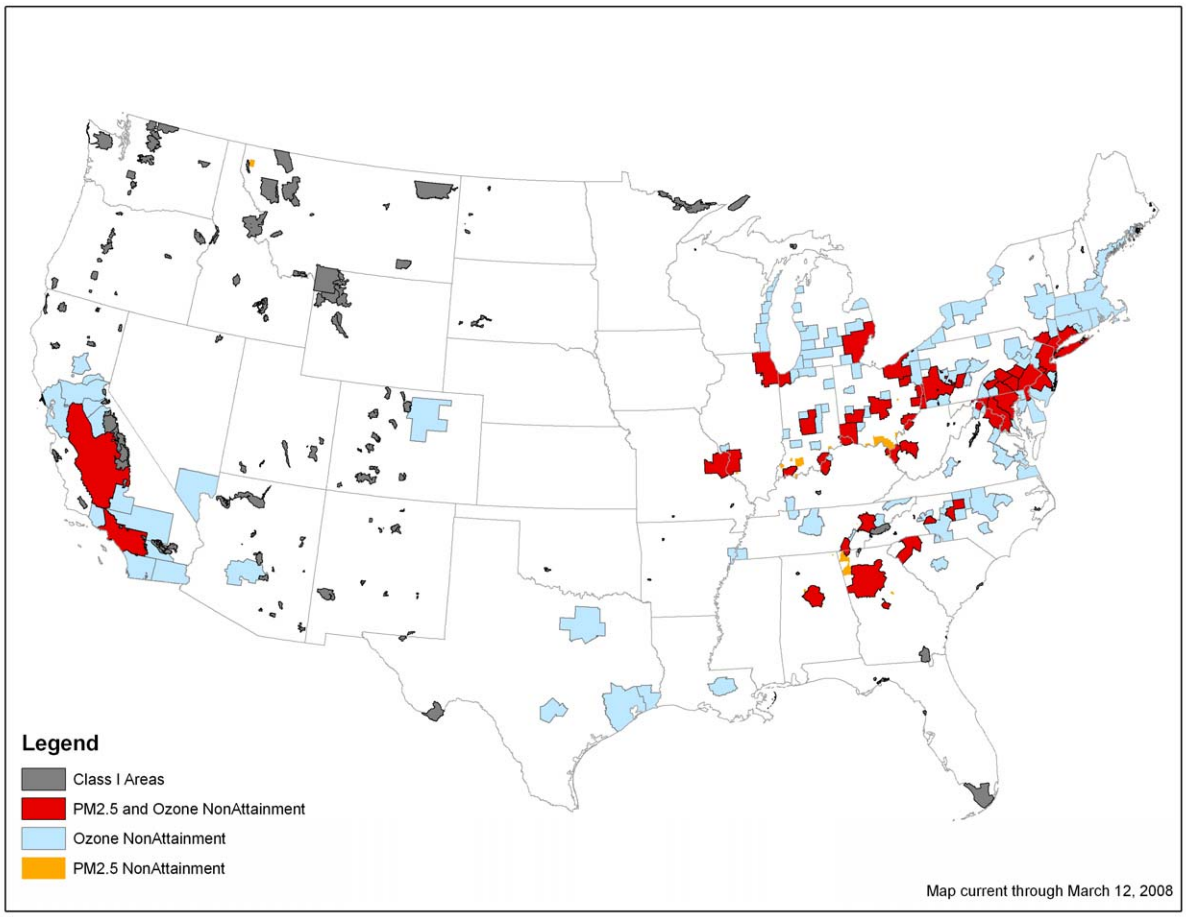

Figure 2-1: 8-Hour Ozone and $PM_{2.5}$ Nonattainment Areas and Mandatory Class I Federal Areas

2.1 Ozone

In this section we review the health and welfare effects of ozone exposure. We also describe the air quality monitoring and modeling data that indicates people in many areas across the country are exposed to levels of ambient ozone above the 1997 and 2008 ozone NAAQS. The data also indicates that in the future people will continue to live in counties with ozone levels above the NAAQS without additional federal, state or local measures. Emissions of volatile organic compounds (VOCs), of which HC are a subset, and NOx from the engines, vessels and equipment subject to this rule contribute to these ozone concentrations. Information on air quality was gathered from a variety of sources, including monitored ozone concentrations, air quality modeling forecasts conducted for this rulemaking, and other state and local air quality information.

2.1.1 Science of Ozone Formation

Ground-level ozone pollution is formed by the reaction of VOCs and NOx in the atmosphere in the presence of heat and sunlight. These pollutants, often referred to as ozone precursors, are emitted by many types of pollution sources such as highway vehicles and nonroad engines (including those subject to this rule), power plants, chemical plants, refineries, makers of consumer and commercial products, industrial facilities, and smaller area sources.

The science of ozone formation, transport, and accumulation is complex.[1] Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions, many of which are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up and result in more ozone than typically would occur on a single high-temperature day. Ozone can be transported hundreds of miles downwind of precursor emissions, resulting in elevated ozone levels even in areas with low VOC or NOx emissions.

The highest levels of ozone are produced when both VOC and NOx emissions are present in significant quantities on clear summer days. Relatively small amounts of NOx enable ozone to form rapidly when VOC levels are relatively high, but ozone production is quickly limited by removal of the NOx. Under these conditions NOx reductions are highly effective in reducing ozone while VOC reductions have little effect. Such conditions are called "NOx-limited". Because the contribution of VOC emissions from biogenic (natural) sources to local ambient ozone concentrations can be significant, even some areas where man-made VOC emissions are relatively low can be NOx-limited.

Ozone concentrations in an area also can be lowered by the reaction of nitric oxide (NO) with ozone, forming nitrogen dioxide (NO_2); as the air moves downwind and the cycle continues, the NO_2 forms additional ozone. The importance of this reaction depends, in part, on the relative concentrations of NOx, VOC, and ozone, all of which change with time and location. When NOx levels are relatively high and VOC levels relatively low, NOx forms inorganic nitrates (i.e., particles) but relatively little ozone. Such conditions are called "VOC-limited". Under these conditions, VOC reductions are effective in reducing ozone, but NOx reductions can actually increase local ozone under certain circumstances. Even in VOC-limited urban areas, NOx reductions are not expected to increase ozone levels if the NOx reductions are sufficiently large.

Rural areas are usually NOx-limited, due to the relatively large amounts of biogenic VOC emissions in such areas. Urban areas can be either VOC- or NOx-limited, or a mixture of both, in which ozone levels exhibit moderate sensitivity to changes in either pollutant.

2.1.2 Health Effects of Ozone Pollution

Exposure to ambient ozone contributes to a wide range of adverse health effects[A].

[A] Human exposure to ozone varies over time due to changes in ambient ozone concentration and because people move between locations which have notable different ozone concentrations. Also, the amount of ozone

These health effects are well documented and are critically assessed in the EPA ozone air quality criteria document (ozone AQCD) and EPA staff paper.[2,3] We are relying on the data and conclusions in the ozone AQCD and staff paper, regarding the health effects associated with ozone exposure.

Ozone-related health effects include lung function decrements, respiratory symptoms, aggravation of asthma, increased hospital and emergency room visits, increased asthma medication usage, and a variety of other respiratory effects. Cell-level effects such as, inflammation of lungs, have been documented as well. In addition, there is suggestive evidence of a contribution of ozone to cardiovascular-related morbidity and highly suggestive evidence that short-term ozone exposure directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality, but additional research is needed to clarify the underlying mechanisms causing these effects. In a recent report on the estimation of ozone-related premature mortality published by the National Research Council (NRC), a panel of experts and reviewers concluded that short-term exposure to ambient ozone is likely to contribute to premature deaths and that ozone-related mortality should be included in estimates of the health benefits of reducing ozone exposure.[4] People who appear to be more susceptible to effects associated with exposure to ozone include children, asthmatics and the elderly. Those with greater exposures to ozone, for instance due to time spent outdoors (e.g., children and outdoor workers), are also of concern.

Based on a large number of scientific studies, EPA has identified several key health effects associated with exposure to levels of ozone found today in many areas of the country. Short-term (1 to 3 hours) and prolonged exposures (6 to 8 hours) to higher ambient ozone concentrations have been linked to lung function decrements, respiratory symptoms, increased hospital admissions and emergency room visits for respiratory problems.[5, 6, 7, 8, 9, 10] Repeated exposure to ozone can increase susceptibility to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.[11, 12, 13, 14, 15] Repeated exposure to sufficient concentrations of ozone can also cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure, which over time could affect premature aging of the lungs and/or the development of chronic respiratory illnesses, such as emphysema and chronic bronchitis.[16, 17, 18, 19]

Children and adults who are outdoors and active during the summer months, such as construction workers, are among those most at risk of elevated ozone exposures.[20] Children and outdoor workers tend to have higher ozone exposure because they typically are active outside, working, playing and exercising, during times of day and seasons (e.g., the summer) when ozone levels are highest.[21] For example, summer camp studies in the Eastern United States and Southeastern Canada have reported statistically significant reductions in lung function in children who are active outdoors.[22, 23, 24, 25, 26, 27, 28, 29] Further, children are more at risk of experiencing health effects from ozone exposure than adults because their respiratory systems are still developing. These individuals (as well as people with respiratory illnesses such as asthma, especially asthmatic children) can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low

delivered to the lung is not only influenced by the ambient concentration but also by the individuals breathing route and rate.

ozone levels during prolonged periods of moderate exertion.[30, 31, 32, 33]

2.1.3 Current Ozone Levels

The small SI and marine SI engine emission reductions will assist ozone nonattainment areas in reaching the standard by each area's respective attainment date and/or assist in maintaining the ozone standard in the future. In this and the following section we present information on current and model-projected future ozone levels.

A nonattainment area is defined in the CAA as an area that is violating a NAAQS or is contributing to a nearby area that is violating the NAAQS. EPA designated nonattainment areas for the 1997 ozone NAAQS in June 2004. The final rule on Air Quality Designations and Classifications for the 1997 Ozone NAAQS (69 FR 23858, April 30, 2004) identifies the criteria that EPA considered in making the 1997 8-hour ozone nonattainment designations, including 2001-2003 measured data, air quality in adjacent areas, and other factors.[B]

As of March 12, 2008 there are approximately 140 million people living in 72 areas designated as nonattainment with the 1997 8-hour ozone NAAQS. There are 337 full or partial counties that make up the 8-hour ozone nonattainment areas. These numbers do not include the people living in areas where there is a future risk of failing to maintain or attain the 8-hour ozone NAAQS. The 1997 8-hour ozone nonattainment areas, nonattainment counties, and populations are listed in Appendix 2A to this RIA.

EPA has recently amended the ozone NAAQS (73 FR 16436, March 27, 2008). The final ozone NAAQS rule addresses revisions to the primary and secondary NAAQS for ozone to provide increased protection of public health and welfare, respectively. With regard to the primary standard for ozone, EPA has revised the level of the 8-hour standard to 0.075 parts per million (ppm), expressed to three decimal places. With regard to the secondary standard for ozone, EPA has revised the current 8-hour standard by making it identical to the revised primary standard.

States with ozone nonattainment areas are required to take action to bring those areas into compliance in the future. The attainment date assigned to an ozone nonattainment area is based on the area's classification. Most ozone nonattainment areas will be required to attain the 1997 8-hour ozone NAAQS in the 2007 to 2013 time frame and then be required to maintain it thereafter.[C] The attainment dates associated with the potential nonattainment areas

[B] An ozone design value is the concentration that determines whether a monitoring site meets the NAAQS for ozone. Because of the way they are defined, design values are determined based on three consecutive-year monitoring periods. For example, an 8-hour ozone design value is the fourth highest daily maximum 8-hour average ozone concentration measured over a three-year period at a given monitor. The full details of these determinations (including accounting for missing values and other complexities) are given in Appendices H and I of 40 CFR Part 50. For a county, the design value is the highest design value from among all the monitors with valid design values within that county. If a county does not contain an ozone monitor, it does not have a design value. However, readers should note that ozone design values generally represent air quality across a broad area and that absence of a design value does not imply that the county is in compliance with the ozone NAAQS.

[C] The Los Angeles South Coast Air Basin 8-hour ozone nonattainment area is designated as severe and will have to attain before June 15, 2021. The South Coast Air Basin has recently applied to be redesignated as an extreme

based on the 2008 8-hour ozone NAAQS will likely be in the 2013 to 2021 timeframe, depending on the severity of the problem. Table 2-1 provides an estimate, based on 2004-06 air quality data, of the counties with design values greater than the 2008 ozone NAAQS. We expect many of the ozone nonattainment areas will need to adopt additional emissions reduction programs to attain and maintain the ozone NAAQS. The expected VOC and NO_x reductions from these standards, which take effect between 2009 and 2013, will be useful to states as they seek to either attain or maintain the ozone NAAQS.

Table 2-1 Counties with Design Values Greater Than the 2008 Ozone NAAQS Based on 2004-2006 Air Quality Data

	Number of Counties	Population[a]
1997 Ozone Standard: counties within the 72 areas currently designated as nonattainment	337	139,633,458
2008 Ozone Standard: additional counties that would not meet the 2008 NAAQS[b]	74	15,984,135
Total	**411**	**155,617,593**

Notes:
[a] Population numbers are from 2000 census data.
[b] Attainment designations for the 2008 ozone NAAQS have not yet been made. Nonattainment for the 2008 Ozone NAAQS will be based on three years of air quality data from later years. Also, the county numbers in the table include only the counties with monitors violating the 2008 Ozone NAAQS. The numbers in this table may be an underestimate of the number of counties and populations that will eventually be included in areas with multiple counties designated nonattainment.

2.1.4 Projected Ozone Levels

In conjunction with this rulemaking, we performed a series of air quality modeling simulations for the continental U.S. The model simulations were performed for several emissions scenarios including the following: 2002 baseline projection, 2020 baseline projection, 2020 baseline projection with small SI/marine SI engine controls, 2030 baseline projection, and 2030 baseline projection with small SI/marine SI engine controls. Information on the air quality modeling methodology is contained in Section 2.3 as well as the air quality modeling technical support document (AQ TSD). In the following sections we describe our modeling of 8-hour ozone levels in the future with and without the controls being finalized in this action.

2.1.4.1 Projected 8-Hour Ozone Levels without this Rulemaking

EPA has already adopted many emission control programs that are expected to reduce ambient ozone levels. These control programs include the Locomotive and Marine Rule (73 FR 25098, May 6, 2008), Clean Air Interstate Rule (70 FR 25162, May 12, 2005), the Clean Air Nonroad Diesel rule (69 FR 38957, June 29, 2004), and the Heavy Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements (66 FR 5002, Jan. 18, 2001). As a result of these programs, the number of areas that continue to violate the 8-hour ozone NAAQS in the future is expected to decrease.

nonattainment area which will make their attainment date June 15, 2024.

The baseline air quality modeling completed for this rule predicts that without additional local, regional or national controls there will continue to be a need for reductions in 8-hour ozone concentrations in some areas in the future. The determination that an area is at risk of exceeding the 8-hour ozone standard in the future was made for all areas with current design values greater than or equal to 85 ppb (or within a 10 percent margin) and with modeling evidence that concentrations at and above these levels will persist into the future.[D] Those interested in greater detail should review the air quality modeling TSD which is included in the docket for this rule.[34]

The baseline inventories that underlie the modeling conducted for this rulemaking include emission reductions from existing federal, state and local controls. There was no attempt to examine the prospects of areas attaining or maintaining the standard with future possible controls. We expect many of the areas to adopt additional emission reduction programs, but we are unable to quantify or rely upon future reductions from additional programs since they have not yet been promulgated. With reductions from programs already in place (but excluding the emission reductions from this rule), the number of counties in 2020 with projected 8-hour ozone design values at or above 85 ppb is expected to be 8 with a population of 22 million people. In addition, in 2020, 37 counties where 27 million people are projected to live, will be within 10 percent of violating the 1997 8-hour ozone NAAQS. The results should therefore be interpreted as indicating counties at risk for violating the ozone NAAQS in the future without additional federal, state or local measures in addition to this rulemaking.

2.1.4.2 Projected 8-Hour Ozone Levels with this Rulemaking

This section summarizes the results of our modeling of ozone air quality impacts in the future due to the reductions in small SI and marine SI emissions finalized in this action. Specifically, we compare baseline scenarios to scenarios with controls. Our modeling indicates that the reductions from this rule will provide nationwide improvements in ambient ozone concentrations and minimize the risk of exposures in future years. Since some of the VOC and NO_x emission reductions from this rule go into effect during the period when some areas are still working to attain the 8-hour ozone NAAQS, the projected emission reductions will assist state and local agencies in their effort to attain the 8-hour ozone standard and help others maintain the standard. Emissions reductions from this rule will also help to counter potential ozone increases due to climate change, which are expected in many urban areas in the United States, but are not reflected in the modeling shown here.[35]

On a population-weighted basis, the average modeled future-year 8-hour ozone design values will decrease by 0.57 ppb in 2020 and 0.76 ppb in 2030. Table 2-2 shows the average change in future year eight-hour ozone design values for: (1) all counties with 2002 baseline design values, (2) counties with baseline design values that exceeded the standard in 2000-2004 (“violating” counties), (3) counties that did not exceed the standard, but were within 10 percent of it in 2000-2004, (4) counties with future year design values that exceeded the

[D] Ozone design values are reported in parts per million (ppm) as specified in 40 CFR Part 50. Due to the scale of the design value changes in this action results have been presented in parts per billion (ppb) format.

standard, and (5) counties with future year design values that did not exceed the standard, but were within 10 percent of it in 2020 and 2030. Counties within ten percent of the standard are intended to reflect counties that meet the standard, but will likely benefit from help in maintaining that status in the face of growth. All of these metrics show a decrease in 2020 and 2030, indicating in five different ways the overall improvement in ozone air quality.

Table 2-2 Average Change in Projected Future Year 8-hour Ozone Design Value as a Result of the Small SI and Marine SI controls

Average[a]	Number of US Counties	Change in 2020 design value[b] (ppb)	Change in 2030 design value[b] (ppb)
All	660	-0.47	-0.66
All, population-weighted	660	-0.57	-0.76
Counties whose base year is violating the 1997 8-hour ozone standard	261	-0.62	-0.88
Counties whose base year is violating the 1997 8-hour ozone standard, population-weighted	261	-0.61	-0.80
Counties whose base year is within 10 percent of the 1997 8-hour ozone standard	223	-0.42	-0.61
Counties whose base year is within 10 percent of the 1997 8-hour ozone standard, population-weighted	223	-0.55	-0.78
Counties whose future year is violating the 1997 8-hour ozone standard	8 (2020) 6 (2030)	-0.13	-0.10
Counties whose future year is violating the 1997 8-hour ozone standard, population-weighted	8 (2020) 6 (2030)	-0.17	-0.13
Counties whose future year is within 10 percent of the 1997 8-hour ozone standard	37 (2020) 23 (2030)	-0.71	-1.05
Counties whose future year is within 10 percent of the 1997 8-hour ozone standard, population-weighted	37 (2020) 23 (2030)	-0.54	-0.79

Notes:

[a] averages are over counties with 2002 modeled design values

[b] Ozone design values are reported in parts per million (ppm) as specified in 40 CFR Part 50. Due to the scale of the design value changes in this action results have been presented in parts per billion (ppb) format.

Table 2-3 lists the counties with projected 8-hour ozone design values that violate or are within 10 percent of the 1997 8-hour ozone standard in 2020after application of the small SI and marine SI controls. Counties are marked with a "V" in the table if their projected design values are greater than or equal to 85 ppb. Counties are marked with an "X" in the table if their projected annual design values are greater than or equal to 76.5 ppb, but less than 85 ppb. The counties marked "X" are not projected to violate the standard, but to be close to it, so the rule will help assure that these counties continue to meet the standard. The current design values are also presented in Table 2-3. Recall that we project future design values only for counties that have current design values, so this list is limited to those counties with ambient monitoring data sufficient to calculate current 3-year design values.

Figure 2-2 illustrates the geographic impact of the small SI and marine SI engine controls on 8-hour ozone design values in 2020. Some of the most significant decreases will occur in the great lakes region, the gulf coast region, the northeast corridor and in the Seattle region. The maximum decreases in a 2020 design values is 2.0 ppb in Cape Cod, Massachusetts.

Table 2-3 Counties with 2020 8-hour Ozone Design Values in Violation or Within 10 percent of the 1997 Ozone Standard as a Result of the Small SI and Marine SI Controls

State	County	2000-2004 Average 8-Hour Ozone DV (ppb)[a]	2020 modeling projections of 8-Hour Ozone DV	2020 Population
CA	El Dorado	105.0	X	236,310
CA	Fresno	110.0	X	1,066,878
CA	Kern	114.3	X	876,131
CA	Kings	95.7	V	173,390
CA	Los Angeles	121.3	X	10,376,013
CA	Madera	91.0	V	173,940
CA	Merced	101.7	V	277,863
CA	Nevada	97.7	V	131,831
CA	Orange	85.3	V	3,900,599
CA	Placer	98.3	V	451,620
CA	Riverside	115.0	X	2,252,510
CA	Sacramento	99.0	V	1,640,590
CA	San Bernardino	128.7	X	2,424,764
CA	San Diego	92.3	V	3,863,460
CA	Stanislaus	95.0	V	607,766
CA	Tulare	105.7	X	477,296
CA	Tuolumne	91.0	V	70,570
CT	Fairfield	98.3	V	962,824
CT	New Haven	98.3	V	898,415
IN	Lake	88.3	V	509,293
LA	East Baton Rouge	87.0	V	522,399
MD	Harford	100.3	V	317,847
NJ	Camden	99.7	V	547,817
NJ	Gloucester	98.0	V	304,105
NJ	Mercer	97.7	V	392,236
NJ	Ocean	105.7	V	644,323
NY	Suffolk	97.0	V	1,598,742
OH	Ashtabula	95.7	V	108,355
OH	Geauga	99.0	V	114,438
PA	Bucks	99.0	V	711,275
PA	Philadelphia	96.7	V	1,394,176
TX	Brazoria	94.0	V	322,385
TX	Harris	102.0	X	4,588,812
TX	Jefferson	91.0	V	272,075
WI	Kenosha	98.3	V	184,825
WI	Racine	91.7	V	212,351
WI	Sheboygan	97.0	V	128,777

Notes:

[a] Ozone design values are reported in parts per million (ppm) as specified in 40 CFR Part 50. Due to the scale of the design value changes in this action results have been presented in parts per billion (ppb) format.

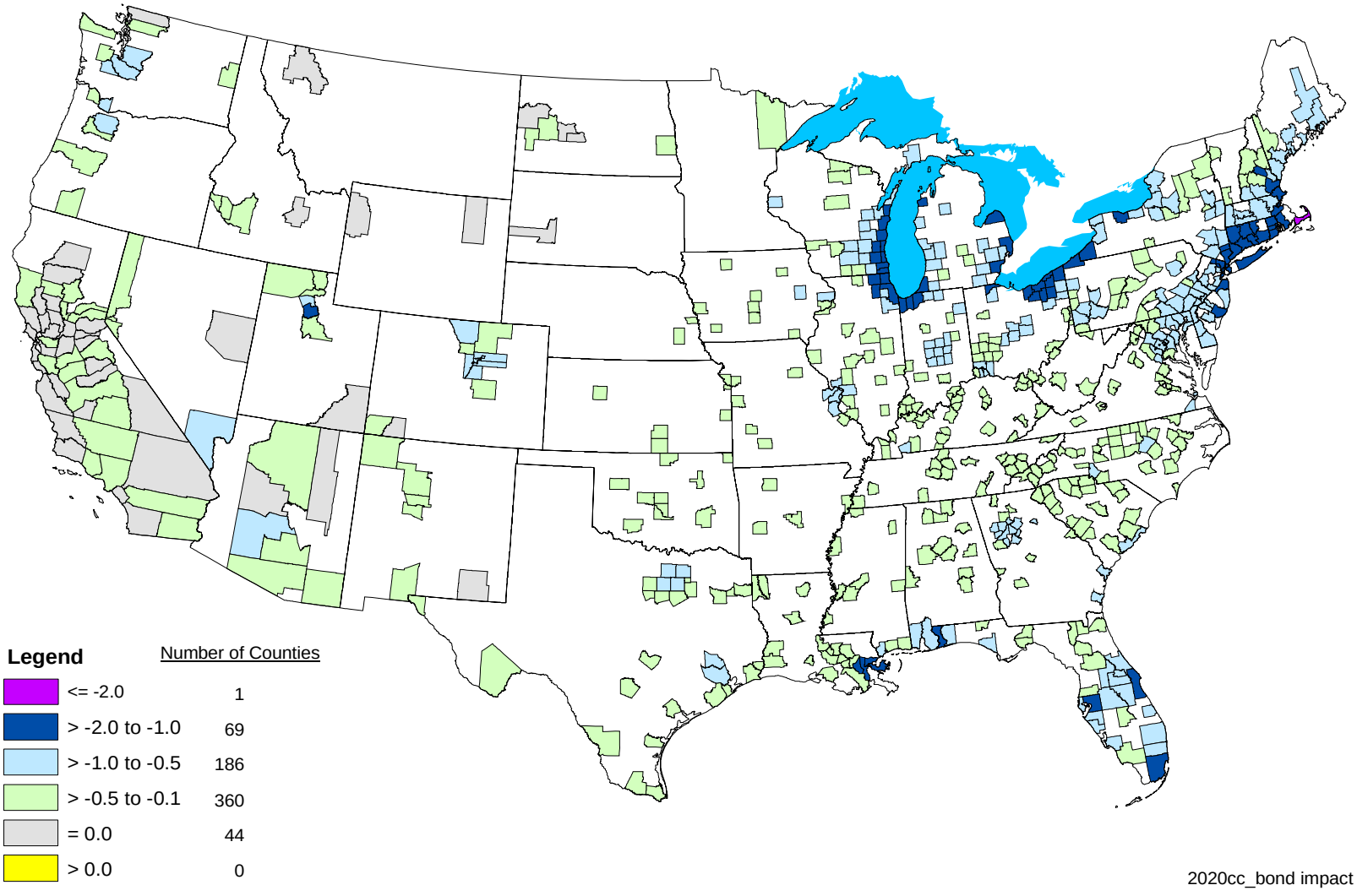

Figure 2-2 Impact of Small SI and Marine SI controls on 8-hour Ozone Design Values in 2020 (units are ppb)

2.1.5 Environmental Effects of Ozone Pollution

There are a number of public welfare effects associated with the presence of ozone in the ambient air.[36] In this section we discuss the impact of ozone on plants, including trees, agronomic crops and urban ornamentals.

2.1.5.1 Impacts on Vegetation

The Air Quality Criteria Document for Ozone and related Photochemical Oxidants notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant. Like carbon dioxide (CO_2) and other gaseous substances, ozone enters plant tissues primarily through apertures (stomata) in leaves in a process called "uptake".[37] Once sufficient levels of ozone, a highly reactive substance, (or its reaction products) reaches the interior of plant cells, it can inhibit or damage essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns.[38,39] This damage is commonly manifested as visible foliar injury such as chlorotic or necrotic spots, increased leaf senescence (accelerated leaf aging) and/or reduced photosynthesis. All these effects reduce a plant's capacity to form carbohydrates, which are the primary form of energy used by plants.[40] With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, more susceptible to disease, insect attack, harsh weather (e.g., drought, frost) and other environmental stresses. Furthermore, there is evidence that ozone can interfere with the formation of mycorrhiza, essential symbiotic fungi associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont.[41,42]

Ozone can produce both acute and chronic injury in sensitive species depending on the concentration level and the duration of the exposure. Ozone effects also tend to accumulate over the growing season of the plant, so that even lower concentrations experienced for a longer duration have the potential to create chronic stress on sensitive vegetation. Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of O_3 uptake through closure of stomata).[43,44,45] Other resistance mechanisms may involve the intercellular production of detoxifying substances. Several biochemical substances capable of detoxifying ozone have been reported to occur in plants including the antioxidants ascorbate and glutathione. After injuries have occurred, plants may be capable of repairing the damage to a limited extent.[46]

Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and response to ozone, it is not possible to identify threshold values above which

ozone is consistently toxic for all plants. The next few paragraphs present additional information on ozone damage to trees, ecosystems, agronomic crops and urban ornamentals.

Ozone also has been conclusively shown to cause discernible injury to forest trees.[47,48] In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts. Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function.[49, 50]

Because plants are at the center of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone impacts at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors.[51] In most instances, responses to chronic or recurrent exposure in forested ecosystems are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems.[52,53,54] It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States.

Laboratory and field experiments have also shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the Unites States."[55] In addition, economic studies have shown reduced economic benefits as a result of predicted reductions in crop yields associated with observed ozone levels.[56, 57, 58]

Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels. It is estimated that more than $20 billion (1990 dollars) are spent annually on landscaping using ornamentals, both by private property owners/tenants and by governmental units responsible for public areas.[59] This is therefore a potentially costly environmental effect. However, in the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative analysis has been conducted.

2.2 Particulate Matter

In this section we review the health and welfare effects of PM. We also describe air quality monitoring and modeling data that indicate many areas across the country continue to be exposed to levels of ambient PM above the NAAQS. Emissions of PM, HCs and NOx from the engines, vessels and equipment subject to this rule contribute to these PM concentrations. Information on air quality was gathered from a variety of sources, including

monitored PM concentrations, air quality modeling forecasts conducted for this rulemaking, and other state and local air quality information.

2.2.1 Science of PM Formation

Particulate matter (PM) represents a broad class of chemically and physically diverse substances. It can be principally characterized as discrete particles that exist in the condensed (liquid or solid) phase spanning several orders of magnitude in size. PM_{10} refers to particles generally less than or equal to 10 micrometers (μm) in aerodynamic diameter. $PM_{2.5}$ refers to fine particles, generally less than or equal to 2.5 μm in aerodynamic diameter. Inhalable (or "thoracic") coarse particles refer to those particles generally greater than 2.5 μm but less than or equal to 10 μm in aerodynamic diameter. Ultrafine PM refers to particles generally less than 100 nanometers (0.1 μm) in aerodynamic diameter. Larger particles (>10 μm) tend to be removed by the respiratory clearance mechanisms, whereas smaller particles are deposited deeper in the lungs.

Fine particles are produced primarily by combustion processes and by transformations of gaseous emissions (e.g., SOx, NOx and VOCs) in the atmosphere. The chemical and physical properties of $PM_{2.5}$ may vary greatly with time, region, meteorology and source category. Thus, $PM_{2.5}$, may include a complex mixture of different pollutants including sulfates, nitrates, organic compounds, elemental carbon and metal compounds. These particles can remain in the atmosphere for days to weeks and travel through the atmosphere hundreds to thousands of kilometers.

Particles span many sizes and shapes and consist of hundreds of different chemicals. Particles are emitted directly from sources and are also formed through atmospheric chemical reactions; the former are often referred to as “primary” particles, and the latter as “secondary” particles. In addition, there are also physical, non-chemical reaction mechanisms that contribute to secondary particles. Particle pollution also varies by time of year and location and is affected by several weather-related factors, such as temperature, clouds, humidity, and wind. A further layer of complexity comes from a particle’s ability to shift between solid/liquid and gaseous phases, which is influenced by concentration, meteorology, and temperature.

2.2.2 Health Effects of PM

As stated in EPA’s Particulate Matter Air Quality Criteria Document (PM AQCD), available scientific findings “demonstrate well that human health outcomes are associated with ambient PM.”[E] We are relying on the data and conclusions in the PM AQCD and PM Staff Paper, which reflects EPA’s analysis of policy-relevant science from the PM AQCD, regarding the health effects associated with particulate matter.[60,61] We also present additional

[E] Personal exposure includes contributions from many different types of particles, from many sources, and in many different environments. Total personal exposure to PM includes both ambient and nonambient components; and both components may contribute to adverse health effects.

recent studies published after the cut-off date for the PM AQCD.[F62] Taken together this information supports the conclusion that PM-related emissions such as those controlled in this action are associated with adverse health effects. Information on PM-related mortality and morbidity is presented first, followed by information on near-roadway exposure studies, marine ports and rail yard exposure studies.

2.2.2.1 Short-term Exposure Mortality and Morbidity Studies

As discussed in the PM AQCD, short-term exposure to $PM_{2.5}$ is associated with mortality from cardiopulmonary diseases (PM AQCD, p. 8-305), hospitalization and emergency department visits for cardiopulmonary diseases (PM AQCD, p. 9-93), increased respiratory symptoms (PM AQCD, p. 9-46), decreased lung function (PM AQCD Table 8-34) and physiological changes or biomarkers for cardiac changes (PM AQCD, Section 8.3.1.3.4). In addition, the PM AQCD describes a limited body of new evidence from epidemiologic studies for potential relationships between short term exposure to PM and health endpoints such as low birth weight, preterm birth, and neonatal and infant mortality. (PM AQCD, Section 8.3.4).

Among the studies of effects from short-term exposure to $PM_{2.5}$, several specifically address the contribution of mobile sources to short-term $PM_{2.5}$ effects on daily mortality. These studies indicate that there are statistically significant associations between mortality and PM related to mobile source emissions (PM AQCD, p.8-85). The analyses incorporate source apportionment tools into daily mortality studies and are briefly mentioned here. Analyses incorporating source apportionment by factor analysis with daily time-series studies of daily death indicated a relationship between mobile source $PM_{2.5}$ and mortality.[63,64] Another recent study in 14 U.S. cities examined the effect of PM_{10} exposures on daily hospital admissions for cardiovascular disease. This study found that the effect of PM_{10} was significantly greater in areas with a larger proportion of PM_{10} coming from motor vehicles, indicating that PM_{10} from these sources may have a greater effect on the toxicity of ambient PM_{10} when compared with other sources.[65] These studies provide evidence that PM-related emissions, specifically from mobile sources, are associated with adverse health effects

Long-term Exposure Mortality and Morbidity Studies

Long-term exposure to elevated ambient $PM_{2.5}$ is associated with mortality from cardiopulmonary diseases and lung cancer (PM AQCD, p. 8-307), and effects on the respiratory system such as decreased lung function or the development of chronic respiratory disease (PM AQCD, pp. 8-313, 8-314). Of specific importance to this rulemaking, the PM AQCD also notes that the PM components of gasoline and diesel engine exhaust represent

[F] These additional studies are included in the 2006 Provisional Assessment of Recent Studies on Health Effects of Particulate Matter Exposure. The provisional assessment did not and could not (given a very short timeframe) undergo the extensive critical review by EPA, CASAC, and the public, as did the PM AQCD. The provisional assessment found that the "new" studies expand the scientific information and provide important insights on the relationship between PM exposure and health effects of PM. The provisional assessment also found that "new" studies generally strengthen the evidence that acute and chronic exposure to fine particles and acute exposure to thoracic coarse particles are associated with health effects.

one class of hypothesized likely important contributors to the observed ambient PM-related increases in lung cancer incidence and mortality (PM AQCD, p. 8-318).

The PM AQCD and PM Staff Paper emphasize the results of two long-term studies, the Six Cities and American Cancer Society (ACS) prospective cohort studies, based on several factors – the inclusion of measured PM data, the fact that the study populations were similar to the general population, and the fact that these studies have undergone extensive reanalysis (PM AQCD, p. 8-306, Staff Paper, p.3-18).[66,67,68] These studies indicate that there are significant associations for all-cause, cardiopulmonary, and lung cancer mortality with long-term exposure to $PM_{2.5}$. One analysis of a subset of the ACS cohort data, which was published after the PM AQCD was finalized but in time for the 2006 Provisional Assessment, found a larger association than had previously been reported between long-term $PM_{2.5}$ exposure and mortality in the Los Angeles area using a new exposure estimation method that accounted for variations in concentration within the city.[69]

As discussed in the PM AQCD, the morbidity studies that combine the features of cross-sectional and cohort studies provide the best evidence for chronic exposure effects. Long-term studies evaluating the effect of ambient PM on children's development have shown some evidence indicating effects of $PM_{2.5}$ and/or PM_{10} on reduced lung function growth (PM AQCD, Section 8.3.3.2.3). In another recent publication included in the 2006 Provisional Assessment, investigators in southern California reported the results of a cross-sectional study of outdoor $PM_{2.5}$ and measures of atherosclerosis in the Los Angeles basin.[70] The study found significant associations between ambient residential $PM_{2.5}$ and carotid intima-media thickness (CIMT), an indicator of subclinical atherosclerosis, an underlying factor in cardiovascular disease.

2.2.2.3 Roadway-Related PM Exposure and Health Studies

A recent body of studies examines traffic-related PM exposures and adverse health effects. These studies are relevant to this rule because highway SI vehicles and nonroad SI engines, vessels and equipment have similar chemical and physical exhaust properties. However, this comparison is qualitative in nature since the near-road environment is influenced by both gasoline (SI) and diesel vehicles, as well as re-entrained road dust and brake and tire wear. One study was done in North Carolina looking at concentrations of $PM_{2.5}$ inside police cars and corresponding physiological changes in the police personnel driving the cars. The authors report significant elevations in markers of cardiac risk associated with concentrations of $PM_{2.5}$ inside police cars on North Carolina state highways.[71] Other studies have found associations between traffic-generated particle concentrations at residences and adverse effects, including all-cause mortality, infant respiratory symptoms, and reduced cognitive functional development.[72,73,74,75] There are other pollutants present in the near roadway environment, including air toxics which are discussed in Section 2.4. Additional information on near-roadway health effects can be found in the recent Mobile Source Air Toxics rule (72 FR 8428, February 26, 2007).

2.2.3 Current and Projected PM Levels

The emission reductions from this rule will assist PM nonattainment areas in reaching the standard by each area's respective attainment date and assist PM maintenance areas in maintaining the PM standards in the future. In this and the following section we present information on current and model-projected future PM levels.

2.2.3.1 Current $PM_{2.5}$ Levels

The small SI and marine SI engine emission reductions will assist PM nonattainment areas in reaching the standard by each area's respective attainment date and/or assist in maintaining the PM standard in the future. In this and the following section we present information on current and model-projected future PM levels.

A nonattainment area is defined in the Clean Air Act (CAA) as an area that is violating an ambient standard or is contributing to a nearby area that is violating the standard. In 2005, EPA designated 39 nonattainment areas for the 1997 $PM_{2.5}$ NAAQS based on air quality design values and a number of other factors (70 FR 943, January 5, 2005; 70 FR 19844, April 14, 2005).[G] These areas are comprised of 208 full or partial counties with a total population exceeding 88 million. The 1997 $PM_{2.5}$ nonattainment counties, areas and populations, as of March 2008, are listed in Appendix 2B to this RIA.

EPA has recently amended the NAAQS for $PM_{2.5}$ (71 FR 61144, October 17, 2006). The final PM NAAQS rule addressed revisions to the primary and secondary NAAQS for $PM_{2.5}$ to provide increased protection of public health and welfare, respectively. The primary $PM_{2.5}$ NAAQS includes a short-term (24-hour) and a long-term (annual) standard. The level of the 24-hour $PM_{2.5}$ NAAQS has been revised from 65 $\mu g/m^3$ to 35 $\mu g/m^3$ to provide increased protection against health effects associated with short-term exposures to fine particles. The current form of the 24-hour $PM_{2.5}$ standard was retained (e.g., based on the 98th percentile concentration averaged over three years). The level of the annual $PM_{2.5}$ NAAQS was retained at 15$\mu g/m^3$, continuing protection against health effects associated with long-term exposures. The current form of the annual $PM_{2.5}$ standard was retained as an annual arithmetic mean averaged over three years, however, the following two aspects of the spatial averaging criteria were narrowed: (1) the annual mean concentration at each site will now be within 10 percent of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair will now yield a correlation coefficient of at least 0.9 for each calendar quarter.

With regard to the secondary standards for $PM_{2.5}$, EPA has revised these standards to be identical in all respects to the revised primary standards. Specifically, EPA has revised the current 24-hour $PM_{2.5}$ secondary standard by making it identical to the revised 24-hour $PM_{2.5}$ primary standard and retained the annual $PM_{2.5}$ secondary standard. This suite of secondary $PM_{2.5}$ standards is intended to provide protection against PM-related public welfare effects, including visibility impairment, effects on vegetation and ecosystems, and material damage and soiling.

[G] The full details involved in calculating a $PM_{2.5}$ design value are given in Appendix N of 40 CFR Part 50.

States with $PM_{2.5}$ nonattainment areas will be required to take action to bring those areas into compliance in the future. Most $PM_{2.5}$ nonattainment areas will be required to attain the 1997 $PM_{2.5}$ NAAQS in the 2010 to 2015 time frame and then be required to maintain the 1997 $PM_{2.5}$ NAAQS thereafter.[H] Nonattainment areas will be designated with respect to the 2006 $PM_{2.5}$ NAAQS in early 2010. The attainment dates associated with the potential nonattainment areas based on the 2006 $PM_{2.5}$ NAAQS will likely be in the 2014 to 2019 timeframe. Table 2-4 provides an estimate, based on 2003-05 air quality data, of the counties with design values greater than the 2006 $PM_{2.5}$ NAAQS. The emission standards being finalized in this action will become effective between 2009 and 2013. The expected $PM_{2.5}$ inventory reductions will be useful to states in attaining or maintaining the $PM_{2.5}$ NAAQS.

Table 2-4 Counties with Design Values Greater Than the 2006 $PM_{2.5}$ NAAQS Based on 2003-2005 Air Quality Data

	Number of Counties	Population[a]
1997 $PM_{2.5}$ Standards: counties within the 39 areas currently designated as nonattainment	208	88,394,000
2006 $PM_{2.5}$ Standards: additional counties that would not meet the 2006 NAAQS[b]	49	18,198,676
Total	**257**	**106,592,676**

Notes:

[a] Population numbers are from 2000 census data.

[b] Attainment designations for the 2006 $PM_{2.5}$ NAAQS have not yet been made. Nonattainment for the 2006 $PM_{2.5}$ NAAQS will be based on three years of air quality data from later years. Also, the county numbers in the table include only the counties with monitors violating the 2006 $PM_{2.5}$ NAAQS. The numbers in this table may be an underestimate of the number of counties and populations that will eventually be included in areas with multiple counties designated nonattainment.

[H] The EPA finalized $PM_{2.5}$ attainment and nonattainment areas in April 2005. The EPA finalized the PM Implementation rule in March 2007.

2.2.3.2 Current PM_{10} Levels

EPA designated PM_{10} nonattainment areas in 1990.[I] As of March 2008, approximately 28 million people live in the 47 areas that are designated as PM_{10} nonattainment, for either failing to meet the PM_{10} NAAQS or for contributing to poor air quality in a nearby area. There are 46 full or partial counties that make up the PM_{10} nonattainment areas.[J]

2.2.3.3 Projected $PM_{2.5}$ Levels

In conjunction with this rulemaking, we performed a series of air quality modeling simulations for the continental U.S. The model simulations were performed for several emissions scenarios including the following: 2002 baseline projection, 2020 baseline projection, 2020 baseline projection with small SI/marine SI engine controls, 2030 baseline projection, and 2030 baseline projection with small SI/marine SI engine controls. Information on the air quality modeling methodology is contained in Section 2.3 as well as the air quality modeling technical support document (AQ TSD). In the following sections we describe projected $PM_{2.5}$ levels in the future with and without the controls being finalized in this action.

2.2.3.2.1 Projected $PM_{2.5}$ Levels without this Rulemaking

Even with the implementation of all current state and federal regulations, including the Locomotive and Marine Rule, CAIR Rule, the NO_x SIP call, nonroad and on-road diesel rules and the Tier 2 rule, there are projected to be U.S. counties violating the $PM_{2.5}$ NAAQS well into the future. The model outputs from the 2002, 2020 and 2030 baselines, combined with current air quality data, were used to identify areas expected to exceed the $PM_{2.5}$ NAAQS in the future.

The baseline air quality modeling conducted for this final rule projects that in 2020, with all current controls in effect, up to 11 counties, with a population of 25 million people, may not attain the annual standard of 15 $\mu g/m^{3.}$ This does not account for additional areas that have air quality measurements within 10 percent of the $PM_{2.5}$ standard. These areas, although not violating the standard, will also benefit from the emissions reductions, ensuring long term maintenance of the PM NAAQS. For example, in 2020, an additional 16 million people are projected to live in 13 counties that have air quality measurements within 10 percent of the 2006 PM NAAQS. This modeling supports the conclusion that there are a substantial number of counties across the US projected to experience $PM_{2.5}$ concentrations at or above the $PM_{2.5}$ NAAQS into the future. Emission reductions from small SI and marine SI engines will be helpful for these counties in attaining and maintaining the $PM_{2.5}$ NAAQS.

[I] A PM_{10} design value is the concentration that determines whether a monitoring site meets the NAAQS for PM_{10}. The full details involved in calculating a PM_{10} design value are given in Appendices H and I of 40 CFR Part 50.

[J] The PM_{10} nonattainment areas are listed in Appendix 2C to this RIA.

2.2.3.2.2 Projected $PM_{2.5}$ Levels With this Rulemaking

The impacts of the small SI and marine SI engine controls were determined by comparing the model results in the future year control runs against the baseline simulations of the same year. On a population-weighted basis, the average modeled future-year annual $PM_{2.5}$ design value (DV) for all counties is expected to decrease by 0.02 $\mu g/m^3$ in 2020 and 2030. There are areas with larger decreases in their future-year annual $PM_{2.5}$ DV, for instance the Chicago region will experience a 0.08 $\mu g/m^3$ reduction by 2030. Figure 2-3 illustrates the geographic impact of the small SI and marine SI engine controls on annual $PM_{2.5}$ design values in 2020.

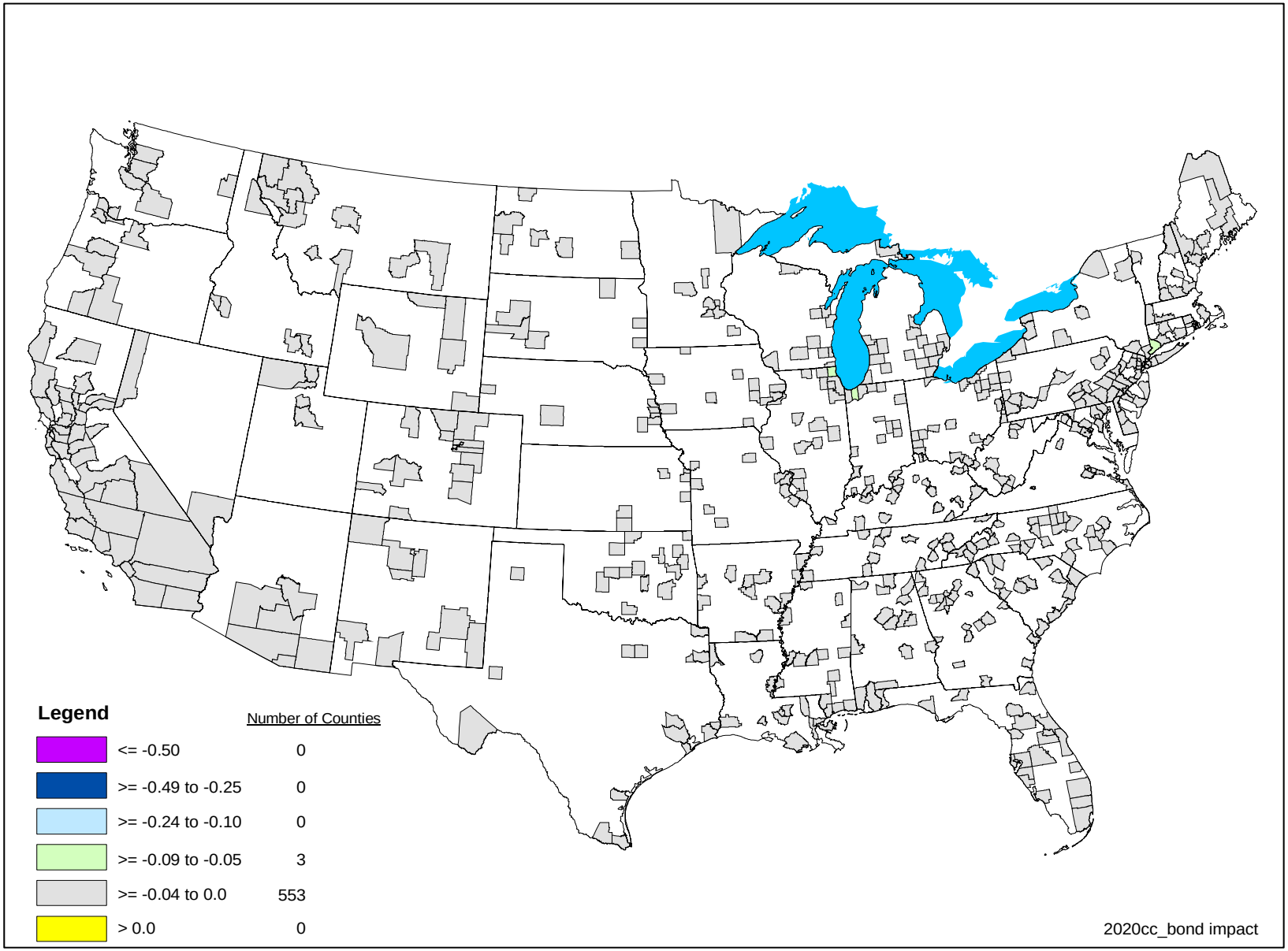

Figure 2-3 Impact of Small SI and Marine SI controls on annual $PM_{2.5}$ Design Values (DV) in 2020 (units are µg/m^3)

Table 2-5 lists the counties with projected annual $PM_{2.5}$ design values that violate or are within 10 percent of the annual $PM_{2.5}$ standard in 2020. Counties are marked with a "V" in the table if their projected design values are greater than or equal to 15.05 $\mu g/m^3$. Counties are marked with an "X" in the table if their projected annual design values are greater than or equal to 13.55 $\mu g/m^3$, but less than 15.05 $\mu g/m^3$. The counties marked "X" are not projected to violate the standard, but to be close to it, so the rule will help assure that these counties continue to meet the standard. The current design values are also presented in Table 2-5. Recall that we project future design values only for counties that have current design values, so this list is limited to those counties with ambient monitoring data sufficient to calculate current 3-year design values.

Table 2-5 Counties with 2020 Projected Annual $PM_{2.5}$ Design Values in Violation or Within 10 percent of the Annual $PM_{2.5}$ Standard as a Result of the Small SI and Marine SI Controls

State	**County**	**2000-2004 Average annual $PM_{2.5}$ DV (ug/m^3)**	**2020 modeling projections of annual $PM_{2.5}$ DV**	**2020 Population**
Alabama	Jefferson Co	18.36	V	681,549
California	Fresno Co	20.02	X	1,066,878
California	Imperial Co	14.44	V	161,555
California	Kern Co	21.77	X	876,131
California	Kings Co	18.77	X	173,390
California	Los Angeles Co	23.16	X	10,376,013
California	Merced Co	16.47	X	277,863
California	Orange Co	18.27	X	3,900,599
California	Riverside Co	27.15	X	2,252,510
California	San Bernardino Co	24.63	X	2,424,764
California	San Diego Co	15.65	V	3,863,460
California	San Joaquin Co	14.84	V	743,469
California	Stanislaus Co	16.49	V	607,766
California	Tulare Co	21.33	X	477,296
Georgia	Fulton Co	18.29	V	929,278
Illinois	Cook Co	17.06	V	5,669,479
Illinois	Madison Co	17.27	V	278,167
Kentucky	Jefferson Co	16.58	V	726,257
Michigan	Wayne Co	19.32	X	1,908,196
Montana	Lincoln Co	15.85	V	20,147
New York	New York Co	17.16	V	1,700,384
Ohio	Cuyahoga Co	18.36	V	1,326,680
Pennsylvania	Allegheny Co	20.99	X	1,242,587
West Virginia	Hancock Co	17.30	V	30,539

2.2.4 Environmental Effects of PM Pollution

In this section we discuss some of the public welfare effects of PM and its precursors, including NOx, such as visibility impairment, atmospheric deposition, and materials damage and soiling.

2.2.4.1 Visibility Impairment

Visibility can be defined as the degree to which the atmosphere is transparent to visible light.[76] Visibility impairment manifests in two principal ways: as local visibility impairment and as regional haze.[77] Local visibility impairment may take the form of a localized plume, a band or layer of discoloration appearing well above the terrain as a result of complex local meteorological conditions. Alternatively, local visibility impairment may manifest as an urban haze, sometimes referred to as a "brown cloud." This urban haze is largely caused by emissions from multiple sources in the urban area and is not typically attributable to only one nearby source or to long-range transport. The second type of visibility impairment, regional haze, usually results from multiple pollution sources spread over a large geographic region. Regional haze can impair visibility over large regions and across states.

Visibility is important because it has direct significance to people's enjoyment of daily activities in all parts of the country. Individuals value good visibility for the well-being it provides them directly, where they live and work and in places where they enjoy recreational opportunities. Visibility is also highly valued in significant natural areas such as national parks and wilderness areas, and special emphasis is given to protecting visibility in these areas.

Fine particles are the major cause of reduced visibility in parts of the United States. To address the welfare effects of PM on visibility, EPA sets secondary $PM_{2.5}$ standards which work in conjunction with the regional haze program. The secondary (welfare-based) $PM_{2.5}$ NAAQS is equal to the suite of primary (health-based) $PM_{2.5}$ NAAQS. The regional haze rule (64 FR 35714, July 1999) was put in place to protect the visibility in mandatory class I federal areas. These areas are defined in Section 162 of the Act as those national parks exceeding 6,000 acres, wilderness areas and memorial parks exceeding 5,000 acres, and all international parks which were in existence on August 7, 1977. A list of the mandatory class I federal areas is included in Appendix 2D. Visibility is impaired in both $PM_{2.5}$ nonattainment areas and mandatory class I federal areas.

Control of small SI and marine SI emissions will improve visibility. The small SI and marine SI engines subject to this rule emit PM and PM precursors and thus contribute to visibility impairment. In the next sections we present current information and projected estimates about visibility impairment related to ambient $PM_{2.5}$ levels across the country and visibility impairment in mandatory class I federal areas. We conclude that visibility will continue to be impaired in the future and the emission reductions from this rule will help improve visibility conditions across the country and in mandatory class I

federal areas. For more information on visibility see the PM AQCD as well as the 2005 PM Staff Paper.[78,79]

2.2.4.1.1 Current Visibility Impairment in $PM_{2.5}$ Nonattainment Areas

As mentioned above, the secondary $PM_{2.5}$ standards were set as equal to the suite of primary $PM_{2.5}$ standards. Almost 90 million people live in the 208 counties that are in nonattainment for the 1997 $PM_{2.5}$ NAAQS, (see Appendix 2A for the complete list of current nonattainment areas). These populations, as well as large numbers of individuals who travel to these areas can experience visibility impairment.

2.2.4.1.2 Current Visibility Impairment at Mandatory Class I Federal Areas

Detailed information about current and historical visibility conditions in mandatory class I federal areas is summarized in the EPA Report to Congress and the 2002 EPA Trends Report.[80,81] The conclusions draw upon the Interagency Monitoring of Protected Visual Environments (IMPROVE) network data. One of the objectives of the IMPROVE monitoring network program is to provide regional haze monitoring representing all mandatory class I federal areas where practical. The National Park Service report also describes the state of national park visibility conditions and discusses the need for improvement.[82]

The regional haze rule requires states to establish goals for each affected mandatory class I federal area that 1) improves visibility on the haziest days (20% most impaired days), 2) ensures no degradation occurs on the cleanest days (20% least impaired days), and 3) achieves natural background visibility levels by 2064. Although there have been general trends toward improved visibility, progress is still needed on the haziest days. Specifically, as discussed in the 2002 EPA Trends Report, without the effects of pollution a natural visual range in the United States is approximately 75 to 150 km in the East and 200 to 300 km in the West. In 2001, the mean visual range for the worst days was 29 km in the East and 98 km in the West.[83]

2.2.4.1.3 Future Visibility Impairment

Additional emission reductions will be needed from a broad set of sources, including those in this action, as part of the overall strategy to achieve the visibility goals of the Act and the regional haze program.

Modeling was used to project visibility conditions in 133 mandatory class I federal areas across the US in 2020 and 2030 as a result of the small SI and marine SI engine standards. The AQ modeling TSD and Section 2.3 of this RIA provide information on the modeling methodology. Table 2-6 below indicates the current monitored deciview values, the natural background levels each area is attempting to reach, and also the projected deciview values in 2020 and 2030 with and without the standards. In 2030, the greatest visibility improvement due to this rule (0.14 deciview) will occur at Brigantine, New Jersey.

Table 2-6 Current (2002) and Future (2020 and 2030) Projected Visibility Conditions With and Without Small SI and Marine SI Rule in Mandatory Class I Federal Areas (20% Worst Days)

Class 1 Area	State	Baseline Visibility	2020 Base	2020 Bond Rule	2030 Base	2030 Bond Rule	Natural Background
Sipsey Wilderness	AL	29.03	23.73	23.72	23.66	23.64	10.99
Caney Creek Wilderness	AR	26.36	22.05	22.03	21.92	21.89	11.58
Upper Buffalo Wilderness	AR	26.27	22.35	22.33	22.19	22.17	11.57
Chiricahua NM	AZ	13.43	13.09	13.09	13.09	13.09	7.21
Chiricahua Wilderness	AZ	13.43	13.09	13.09	13.09	13.09	7.21
Galiuro Wilderness	AZ	13.43	13.07	13.06	13.09	13.09	7.21
Grand Canyon NP	AZ	11.66	11.09	11.09	11.08	11.08	7.14
Mazatzal Wilderness	AZ	13.35	12.72	12.71	12.73	12.71	6.68
Petrified Forest NP	AZ	13.21	12.83	12.82	12.75	12.75	6.49
Pine Mountain Wilderness	AZ	13.35	12.58	12.56	12.54	12.53	6.68
Saguaro NM	AZ	14.83	14.47	14.48	14.44	14.45	6.46
Sierra Ancha Wilderness	AZ	13.67	13.20	13.20	13.15	13.14	6.59
Sycamore Canyon Wilderness	AZ	15.25	14.94	14.93	14.93	14.93	6.69
Agua Tibia Wilderness	CA	23.50	21.14	21.13	20.94	20.94	7.64
Caribou Wilderness	CA	14.15	13.60	13.60	13.51	13.51	7.31
Cucamonga Wilderness	CA	19.94	17.36	17.38	17.10	17.10	7.06
Desolation Wilderness	CA	12.63	12.13	12.13	12.12	12.12	6.12
Dome Land Wilderness	CA	19.43	18.34	18.34	18.11	18.11	7.46
Emigrant Wilderness	CA	17.63	17.21	17.20	17.19	17.19	7.64
Hoover Wilderness	CA	12.87	12.72	12.72	12.74	12.74	7.91
Joshua Tree NM	CA	19.62	17.93	17.97	17.71	17.72	7.19
Lassen Volcanic NP	CA	14.15	13.54	13.54	13.43	13.43	7.31
Lava Beds NM	CA	15.05	14.42	14.42	14.32	14.32	7.86
Mokelumne Wilderness	CA	12.63	12.30	12.30	12.31	12.30	6.12
Pinnacles NM	CA	18.46	17.36	17.34	17.09	17.09	7.99
Point Reyes NS	CA	22.81	21.99	21.98	21.79	21.79	15.77
Redwood NP	CA	18.45	17.86	17.86	17.79	17.78	13.91
San Gabriel Wilderness	CA	19.94	17.25	17.25	16.93	16.93	7.06
San Gorgonio Wilderness	CA	22.17	20.22	20.24	19.70	19.71	7.30
San Jacinto Wilderness	CA	22.17	19.87	19.90	19.55	19.52	7.30
South Warner Wilderness	CA	15.05	14.59	14.59	14.52	14.52	7.86
Thousand Lakes Wilderness	CA	14.15	13.52	13.52	13.41	13.40	7.31
Ventana Wilderness	CA	18.46	17.64	17.63	17.62	17.62	7.99
Yosemite NP	CA	17.63	17.14	17.14	17.11	17.11	7.64
Black Canyon of the Gunnison NM	CO	10.33	9.79	9.79	9.77	9.77	6.24
Eagles Nest Wilderness	CO	9.61	9.03	9.03	8.96	8.95	6.54
Flat Tops Wilderness	CO	9.61	9.25	9.25	9.24	9.24	6.54
Great Sand Dunes NM	CO	12.78	12.35	12.35	12.34	12.34	6.66

Class 1 Area	State	Baseline Visibility	2020 Base	2020 Bond Rule	2030 Base	2030 Bond Rule	Natural Background
La Garita Wilderness	CO	10.33	9.89	9.89	9.88	9.87	6.24
Maroon Bells-Snowmass Wilderness	CO	9.61	9.21	9.21	9.20	9.20	6.54
Mesa Verde NP	CO	13.03	12.39	12.39	12.37	12.37	6.83
Mount Zirkel Wilderness	CO	10.52	10.05	10.05	10.04	10.03	6.44
Rawah Wilderness	CO	10.52	10.04	10.03	10.04	10.02	6.44
Rocky Mountain NP	CO	13.83	13.08	13.06	13.01	12.99	7.24
Weminuche Wilderness	CO	10.33	9.85	9.85	9.85	9.84	6.24
West Elk Wilderness	CO	9.61	9.15	9.15	9.14	9.14	6.54
Chassahowitzka	FL	26.09	21.94	21.92	21.91	21.88	11.21
Everglades NP	FL	22.30	19.77	19.76	19.94	19.91	12.15
St. Marks	FL	26.03	21.82	21.81	21.83	21.81	11.53
Cohutta Wilderness	GA	30.30	23.33	23.32	23.28	23.26	11.14
Okefenokee	GA	27.13	23.42	23.41	23.40	23.39	11.44
Wolf Island	GA	27.13	23.37	23.35	23.32	23.29	11.44
Craters of the Moon NM	ID	14.00	12.97	12.96	12.82	12.80	7.53
Sawtooth Wilderness	ID	13.78	13.63	13.63	13.63	13.63	6.43
Mammoth Cave NP	KY	31.37	25.48	25.47	25.44	25.42	11.08
Acadia NP	ME	22.89	19.77	19.75	19.81	19.78	12.43
Moosehorn	ME	21.72	18.63	18.62	18.64	18.62	12.01
Roosevelt Campobello International Park	ME	21.72	18.45	18.44	18.47	18.45	12.01
Isle Royale NP	MI	20.74	19.10	19.08	19.04	19.01	12.37
Seney	MI	24.16	21.72	21.70	21.66	21.63	12.65
Voyageurs NP	MN	19.27	17.58	17.56	17.43	17.41	12.06
Hercules-Glades Wilderness	MO	26.75	22.93	22.92	22.81	22.78	11.30
Anaconda-Pintler Wilderness	MT	13.41	13.14	13.13	13.11	13.11	7.43
Bob Marshall Wilderness	MT	14.48	14.13	14.12	14.08	14.07	7.74
Cabinet Mountains Wilderness	MT	14.09	13.54	13.53	13.46	13.44	7.53
Gates of the Mountains Wilderness	MT	11.29	10.91	10.91	10.87	10.86	6.45
Medicine Lake	MT	17.72	16.19	16.19	16.09	16.09	7.90
Mission Mountains Wilderness	MT	14.48	14.04	14.04	13.99	13.99	7.74
Scapegoat Wilderness	MT	14.48	14.16	14.15	14.12	14.11	7.74
Selway-Bitterroot Wilderness	MT	13.41	13.04	13.04	12.99	12.99	7.43
UL Bend	MT	15.14	14.64	14.63	14.58	14.57	8.16
Linville Gorge Wilderness	NC	28.77	22.45	22.44	22.41	22.39	11.22
Swanquarter	NC	25.49	21.15	21.11	21.15	21.10	11.94
Lostwood	ND	19.57	17.70	17.70	17.60	17.60	8.00
Theodore Roosevelt NP	ND	17.74	16.49	16.48	16.34	16.34	7.79

Class 1 Area	State	Baseline Visibility	2020 Base	2020 Bond Rule	2030 Base	2030 Bond Rule	Natural Background
Great Gulf Wilderness	NH	22.82	19.45	19.43	19.46	19.43	11.99
Presidential Range-Dry River Wilderness	NH	22.82	19.45	19.43	19.46	19.43	11.99
Brigantine	NJ	29.01	24.85	24.75	24.91	24.77	12.24
Bandelier NM	NM	12.22	11.35	11.35	11.29	11.28	6.26
Bosque del Apache	NM	13.80	12.85	12.85	12.73	12.73	6.73
Gila Wilderness	NM	13.11	12.54	12.54	12.54	12.53	6.69
Pecos Wilderness	NM	10.41	9.97	9.97	9.97	9.97	6.44
Salt Creek	NM	18.03	16.59	16.58	16.52	16.52	6.81
San Pedro Parks Wilderness	NM	10.17	9.43	9.43	9.40	9.40	6.08
Wheeler Peak Wilderness	NM	10.41	9.88	9.88	9.87	9.87	6.44
White Mountain Wilderness	NM	13.70	12.88	12.89	12.87	12.86	6.86
Jarbidge Wilderness	NV	12.07	11.86	11.85	11.85	11.85	7.87
Wichita Mountains	OK	23.81	20.62	20.60	20.55	20.53	7.53
Crater Lake NP	OR	13.74	13.27	13.25	13.20	13.18	7.84
Diamond Peak Wilderness	OR	13.74	13.20	13.19	13.12	13.11	7.84
Eagle Cap Wilderness	OR	18.57	17.83	17.82	17.71	17.70	8.92
Gearhart Mountain Wilderness	OR	13.74	13.37	13.37	13.33	13.33	7.84
Hells Canyon Wilderness	OR	18.55	17.20	17.19	17.04	17.01	8.32
Kalmiopsis Wilderness	OR	15.51	14.98	14.97	14.93	14.92	9.44
Mount Hood Wilderness	OR	14.86	14.13	14.12	14.14	14.12	8.44
Mount Jefferson Wilderness	OR	15.33	14.77	14.76	14.76	14.75	8.79
Mount Washington Wilderness	OR	15.33	14.75	14.74	14.72	14.71	8.79
Mountain Lakes Wilderness	OR	13.74	13.24	13.23	13.17	13.16	7.84
Strawberry Mountain Wilderness	OR	18.57	17.73	17.72	17.60	17.59	8.92
Three Sisters Wilderness	OR	15.33	14.82	14.81	14.79	14.78	8.79
Cape Romain	SC	26.48	22.74	22.72	22.71	22.68	12.12
Badlands NP	SD	17.14	15.84	15.83	15.74	15.74	8.06
Wind Cave NP	SD	15.84	14.91	14.91	14.87	14.86	7.71
Great Smoky Mountains NP	TN	30.28	23.93	23.92	23.86	23.85	11.24
Joyce-Kilmer-Slickrock Wilderness	TN	30.28	23.43	23.42	23.37	23.35	11.24
Big Bend NP	TX	17.30	16.13	16.13	16.15	16.14	7.16
Carlsbad Caverns NP	TX	17.19	15.89	15.89	15.87	15.87	6.68
Guadalupe Mountains NP	TX	17.19	15.87	15.86	15.84	15.84	6.68
Arches NP	UT	11.24	11.11	11.11	11.03	11.01	6.43
Bryce Canyon NP	UT	11.65	11.34	11.34	11.31	11.31	6.86

Class 1 Area	State	Baseline Visibility	2020 Base	2020 Bond Rule	2030 Base	2030 Bond Rule	Natural Background
Canyonlands NP	UT	11.24	10.81	10.81	10.82	10.85	6.43
Zion NP	UT	13.24	12.92	12.95	12.81	12.83	6.99
James River Face Wilderness	VA	29.12	23.34	23.31	23.26	23.23	11.13
Shenandoah NP	VA	29.31	22.80	22.78	22.76	22.73	11.35
Lye Brook Wilderness	VT	24.45	21.08	21.06	21.11	21.08	11.73
Alpine Lake Wilderness	WA	17.84	16.71	16.69	16.60	16.57	8.43
Glacier Peak Wilderness	WA	13.96	13.60	13.60	13.67	13.66	8.01
Goat Rocks Wilderness	WA	12.76	12.05	12.03	12.03	12.02	8.36
Mount Adams Wilderness	WA	12.76	12.01	12.00	11.97	11.96	8.36
Mount Rainier NP	WA	18.24	17.24	17.23	17.21	17.18	8.55
North Cascades NP	WA	13.96	13.57	13.57	13.67	13.66	8.01
Olympic NP	WA	16.74	15.82	15.82	15.89	15.86	8.44
Pasayten Wilderness	WA	15.23	14.84	14.84	14.81	14.81	8.26
Dolly Sods Wilderness	WV	29.04	22.35	22.34	22.33	22.31	10.39
Otter Creek Wilderness	WV	29.04	22.29	22.28	22.27	22.25	10.39
Bridger Wilderness	WY	11.12	10.80	10.80	10.79	10.78	6.58
Fitzpatrick Wilderness	WY	11.12	10.85	10.85	10.84	10.84	6.58
Grand Teton NP	WY	11.76	11.35	11.35	11.31	11.31	6.51
North Absaroka Wilderness	WY	11.45	11.16	11.16	11.13	11.12	6.86
Red Rock Lakes	WY	11.76	11.43	11.42	11.39	11.39	6.51
Teton Wilderness	WY	11.76	11.40	11.39	11.36	11.36	6.51
Washakie Wilderness	WY	11.45	11.17	11.16	11.14	11.14	6.86
Yellowstone NP	WY	11.76	11.38	11.37	11.34	11.33	6.51

[a] The level of visibility impairment in an area is based on the light-extinction coefficient and a unitless visibility index, called a "deciview", which is used in the valuation of visibility. The deciview metric provides a scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. The higher the deciview value, the worse the visibility. Thus, an improvement in visibility is a decrease in deciview value.

2.2.4.2 Particulate Matter Deposition

Particulate matter contributes to adverse effects on vegetation and ecosystems, and to soiling and materials damage. These welfare effects result predominately from exposure to excess amounts of specific chemical species, regardless of their source or predominant form (particle, gas or liquid). Reflecting this fact, the PM AQCD concludes that regardless of size fractions, particles containing nitrates and sulfates have the greatest potential for widespread environmental significance, while effects are also related to other chemical constituents found in ambient PM, such as trace metals and organics. The following characterizations of the nature of these welfare effects are based on the information contained in the PM AQCD and PM Staff Paper.

2.2.4.2.1 Deposition of Nitrates and Sulfates

At current ambient levels, risks to vegetation from short-term exposures to dry deposited particulate nitrate or sulfate are low. However, when found in acid or acidifying deposition, such particles do have the potential to cause direct leaf injury. Specifically, the responses of forest trees to acid precipitation (rain, snow) include accelerated weathering of leaf cuticular surfaces, increased permeability of leaf surfaces to toxic materials, water, and disease agents; increased leaching of nutrients from foliage; and altered reproductive processes—all which serve to weaken trees so that they are more susceptible to other stresses (e.g., extreme weather, pests, pathogens). Acid deposition with levels of acidity associated with the leaf effects described above are currently found in some locations in the eastern U.S.[84] Even higher concentrations of acidity can be present in occult depositions (e.g., fog, mist or clouds) which more frequently impacts higher elevations. Thus, the risk of leaf injury occurring from acid deposition in some areas of the eastern U.S**.** is high. Nitrogen deposition has also been shown to impact ecosystems in the western U.S. A study conducted in the Columbia River Gorge National Scenic Area (CRGNSA), located along a portion of the Oregon/Washington border, indicates that lichen communities in the CRGNSA have shifted to a higher proportion of nitrophilous species and the nitrogen content of lichen tissue is elevated.[85] Lichens are sensitive indicators of nitrogen deposition effects to terrestrial ecosystems and the lichen studies in the Columbia River Gorge clearly show that ecological effects from air pollution are occurring.

Some of the most significant detrimental effects associated with excess reactive nitrogen deposition are those associated with a syndrome known as nitrogen saturation. These effects include: (1) decreased productivity, increased mortality, and/or shifts in plant community composition, often leading to decreased biodiversity in many natural habitats wherever atmospheric reactive nitrogen deposition increases significantly and critical thresholds are exceeded; (2) leaching of excess nitrate and associated base cations from soils into streams, lakes, and rivers, and mobilization of soil aluminum; and (3) fluctuation of ecosystem processes such as nutrient and energy cycles through changes in the functioning and species composition of beneficial soil organisms.[86]

In the U.S. numerous forests now show severe symptoms of nitrogen saturation. These forests include: the northern hardwoods and mixed conifer forests in the Adirondack and Catskill Mountains of New York; the red spruce forests at Whitetop Mountain, Virginia, and Great Smoky Mountains National Park, North Carolina; mixed hardwood watersheds at Fernow Experimental Forest in West Virginia; American beech forests in Great Smoky Mountains National Park, Tennessee; mixed conifer forests and chaparral watersheds in southern California and the southwestern Sierra Nevada in Central California; the alpine tundra/subalpine conifer forests of the Colorado Front Range; and red alder forests in the Cascade Mountains in Washington.

Excess nutrient inputs into aquatic ecosystems (i.e. streams, rivers, lakes, estuaries or oceans) either from direct atmospheric deposition, surface runoff, or leaching from nitrogen saturated soils into ground or surface waters can contribute to conditions of

severe water oxygen depletion; eutrophication and algae blooms; altered fish distributions, catches, and physiological states; loss of biodiversity; habitat degradation; and increases in the incidence of disease.

Severe and persistent eutrophication often directly impacts human activities. For example, losses in the nation's fishery resources may be directly caused by fish kills associated with low dissolved oxygen and toxic blooms. Declines in tourism occur when low dissolved oxygen causes noxious smells and floating mats of algal blooms create unfavorable aesthetic conditions. Risks to human health increase when the toxins from algal blooms accumulate in edible fish and shellfish, and when toxins become airborne, causing respiratory problems due to inhalation. According to a NOAA report, more than half of the nation's estuaries have moderate to high expressions of at least one of these symptoms – an indication that eutrophication is well developed in more than half of U.S. estuaries. [87]

2.2.4.2.2 Deposition of Heavy Metals

Heavy metals, including cadmium, copper, lead, chromium, mercury, nickel and zinc, have the greatest potential for influencing forest growth (PM AQCD, p. 4-87).[88] Investigation of trace metals near roadways and industrial facilities indicate that a substantial load of heavy metals can accumulate on vegetative surfaces. Copper, zinc, and nickel have been documented to cause direct toxicity to vegetation under field conditions (PM AQCD, p. 4-75). Little research has been conducted on the effects associated with mixtures of contaminants found in ambient PM. While metals typically exhibit low solubility, limiting their bioavailability and direct toxicity, chemical transformations of metal compounds occur in the environment, particularly in the presence of acidic or other oxidizing species. These chemical changes influence the mobility and toxicity of metals in the environment. Once taken up into plant tissue, a metal compound can undergo chemical changes, accumulate and be passed along to herbivores or can re-enter the soil and further cycle in the environment. Although there has been no direct evidence of a physiological association between tree injury and heavy metal exposures, heavy metals have been implicated because of similarities between metal deposition patterns and forest decline (PM AQCD, p. 4-76). This hypothesized relationship/correlation was further explored in high elevation forests in the northeastern U.S. These studies measured levels of a group of intracellular compounds found in plants that bind with metals and are produced by plants as a response to sublethal concentrations of heavy metals. These studies indicated a systematic and significant increase in concentrations of these compounds associated with the extent of tree injury. These data strongly imply that metal stress causes tree injury and contributes to forest decline in the northeastern United States (PM AQCD 4-76,77).[89] Contamination of plant leaves by heavy metals can lead to elevated soil levels. Trace metals absorbed into the plant frequently bind to the leaf tissue, and then are lost when the leaf drops (PM AQCD, p. 4-75). As the fallen leaves decompose, the heavy metals are transferred into the soil.[90,91]

The environmental sources and cycling of mercury are currently of particular concern due to the bioaccumulation and biomagnification of this metal in aquatic

ecosystems and the potent toxic nature of mercury in the forms in which is it ingested by people and other animals. Mercury is unusual compared with other metals in that it largely partitions into the gas phase (in elemental form), and therefore has a longer residence time in the atmosphere than a metal found predominantly in the particle phase. This property enables mercury to travel far from the primary source before being deposited and accumulating in the aquatic ecosystem. The major source of mercury in the Great Lakes is from atmospheric deposition, accounting for approximately eighty percent of the mercury in Lake Michigan.[92,93] Over fifty percent of the mercury in the Chesapeake Bay has been attributed to atmospheric deposition.[94] Overall, the National Science and Technology Council identifies atmospheric deposition as the primary source of mercury to aquatic systems.[95] Forty-four states have issued health advisories for the consumption of fish contaminated by mercury; however, most of these advisories are issued in areas without a mercury point source.

Elevated levels of zinc and lead have been identified in streambed sediments, and these elevated levels have been correlated with population density and motor vehicle use.[96,97] Zinc and nickel have also been identified in urban water and soils. In addition, platinum, palladium, and rhodium, metals found in the catalysts of modern motor vehicles, have been measured at elevated levels along roadsides.[98] Plant uptake of platinum has been observed at these locations.

2.2.4.2.3 Deposition of Polycyclic Organic Matter

Polycyclic organic matter (POM) is a byproduct of incomplete combustion and consists of organic compounds with more than one benzene ring and a boiling point greater than or equal to 100 degrees centigrade.[99] Polycyclic aromatic hydrocarbons (PAHs) are a class of POM that contains compounds which are known or suspected carcinogens.

Major sources of PAHs include mobile sources. PAHs in the environment may be present as a gas or adsorbed onto airborne particulate matter. Since the majority of PAHs are adsorbed onto particles less than 1.0 μm in diameter, long range transport is possible. However, studies have shown that PAH compounds adsorbed onto diesel exhaust particulate and exposed to ozone have half lives of 0.5 to 1.0 hours.[100]

Since PAHs are insoluble, the compounds generally are particle reactive and accumulate in sediments. Atmospheric deposition of particles is believed to be the major source of PAHs to the sediments of Lake Michigan.[101,102] Analyses of PAH deposition in Chesapeake and Galveston Bay indicate that dry deposition and gas exchange from the atmosphere to the surface water predominate.[103,104] Sediment concentrations of PAHs are high enough in some segments of Tampa Bay to pose an environmental health threat. EPA funded a study to better characterize the sources and loading rates for PAHs into Tampa Bay.[105] PAHs that enter a water body through gas exchange likely partition into organic rich particles and can be biologically recycled, while dry deposition of aerosols containing PAHs tend to be more resistant to biological recycling.[106] Thus, dry deposition is likely the main pathway for PAH concentrations in sediments while gas/water exchange at the surface may lead to PAH distribution into the food web,

leading to increased health risk concerns.

Trends in PAH deposition levels are difficult to discern because of highly variable ambient air concentrations, lack of consistency in monitoring methods, and the significant influence of local sources on deposition levels.[107] Van Metre et al. noted PAH concentrations in urban reservoir sediments have increased by 200-300% over the last forty years and correlate with increases in automobile use.[108]

Cousins et al. estimate that more than ninety percent of semi-volatile organic compound (SVOC) emissions in the United Kingdom deposit on soil.[109] An analysis of PAH concentrations near a Czechoslovakian roadway indicated that concentrations were thirty times greater than background.[110]

2.2.4.2.4 Materials Damage and Soiling

The effects of the deposition of atmospheric pollution, including ambient PM, on materials are related to both physical damage and impaired aesthetic qualities. The deposition of PM (especially sulfates and nitrates) can physically affect materials, adding to the effects of natural weathering processes, by potentially promoting or accelerating the corrosion of metals, by degrading paints, and by deteriorating building materials such as concrete and limestone. Only chemically active fine particles or hygroscopic coarse particles contribute to these physical effects. In addition, the deposition of ambient PM can reduce the aesthetic appeal of buildings and culturally important articles through soiling. Particles consisting primarily of carbonaceous compounds cause soiling of commonly used building materials and culturally important items such as statues and works of art.

2.3 Air Quality Modeling Methodology

In this section we present information on the air quality modeling, including the model domain and modeling inputs. Further discussion of the modeling methodology, including evaluations of model performance, is included in the Air Quality Modeling Technical Support Document (AQM TSD).[111]

2.3.1 Air Quality Modeling Overview

A national scale air quality modeling analysis was performed to estimate future year 8-hour ozone concentrations, annual $PM_{2.5}$ concentrations, and visibility levels. These projections were used as inputs to the calculation of expected benefits from the small SI and marine SI emissions controls considered in this assessment. The 2002-based CMAQ modeling platform was used as the tool for the air quality modeling of future baseline emissions and control scenarios. It should be noted that the 2002-based modeling platform has recently been finalized and the 2001-based modeling platform was used as the tool for the air quality modeling performed for the proposal. In the next paragraph we discuss some of the differences between the 2001-based platform used for the proposal and the 2002-based platform used for this final rule.

The 2002-based modeling platform includes a number of updates and improvements to data and tools compared to the 2001-based platform that was used for the proposal modeling. For the final rule modeling we used the new 2002 National Emissions Inventory along with updated versions of the models used to project future emissions from electric generating units (EGUs) and onroad and nonroad vehicles. The proposal modeling was based on the 2001 National Emissions Inventory. The new platform also includes 2002 meteorology and more recent ambient design values which were used as the starting point for projecting future air quality. For proposal, we used meteorology for 2001 for modeling the East and 2002 for modeling the West. The updates to CMAQ between proposal and final include (1) an in-cloud sulfate chemistry module that accounts for the nonlinear sensitivity of sulfate formation to varying pH; (2) improved vertical convective mixing; (3) heterogeneous reaction involving nitrate formation; (4) an updated gas-phase chemistry mechanism, Carbon Bond 2005 (CB05); and (5) an aqueous chemistry mechanism that provides a comprehensive simulation of aerosol precursor oxidants.

The CMAQ model is a three-dimensional grid-based Eulerian air quality model designed to estimate the formation and fate of oxidant precursors, primary and secondary particulate matter concentrations and deposition over regional and urban spatial scales (e.g., over the contiguous U.S.).[112,113,114] Consideration of the different processes that affect primary (directly emitted) and secondary (formed by atmospheric processes) PM at the regional scale in different locations is fundamental to understanding and assessing the effects of pollution control measures that affect PM, ozone and deposition of pollutants to the surface. In addition to the CMAQ model, the modeling platform includes the emissions, meteorology, and initial/boundary condition data which are inputs to this model.

The CMAQ model was peer-reviewed in 2003 for EPA as reported in "Peer Review of CMAQ Model".[115] The latest version of CMAQ (Version 4.6.1) was employed for this modeling analysis. This version reflects updates, as mentioned above, in a number of areas to improve the underlying science which include (1) use of a state-of-the science inorganic and organic aerosol module, (2) an in-cloud sulfate chemistry module that accounts for the nonlinear sensitivity of sulfate formation to varying pH, (3) improved vertical convective mixing, (4) heterogeneous reaction involving nitrate formation and (5) an updated Carbon Bond 05 (CB05) gas-phase chemistry mechanism and aqueous chemistry mechanism that provides a comprehensive simulation of aerosol precursor oxidants.

2.3.2 Model Domain and Configuration

The CMAQ modeling domain encompasses all of the lower 48 States and portions of Canada and Mexico. The modeling domain is made up of a large continental U.S. 36 km grid and two 12 km grids (an Eastern US and a Western US domain), as shown in Figure 2-4. The modeling domain contains 14 vertical layers with the top of the modeling domain at about 16,200 meters, or 100 millibars (mb).

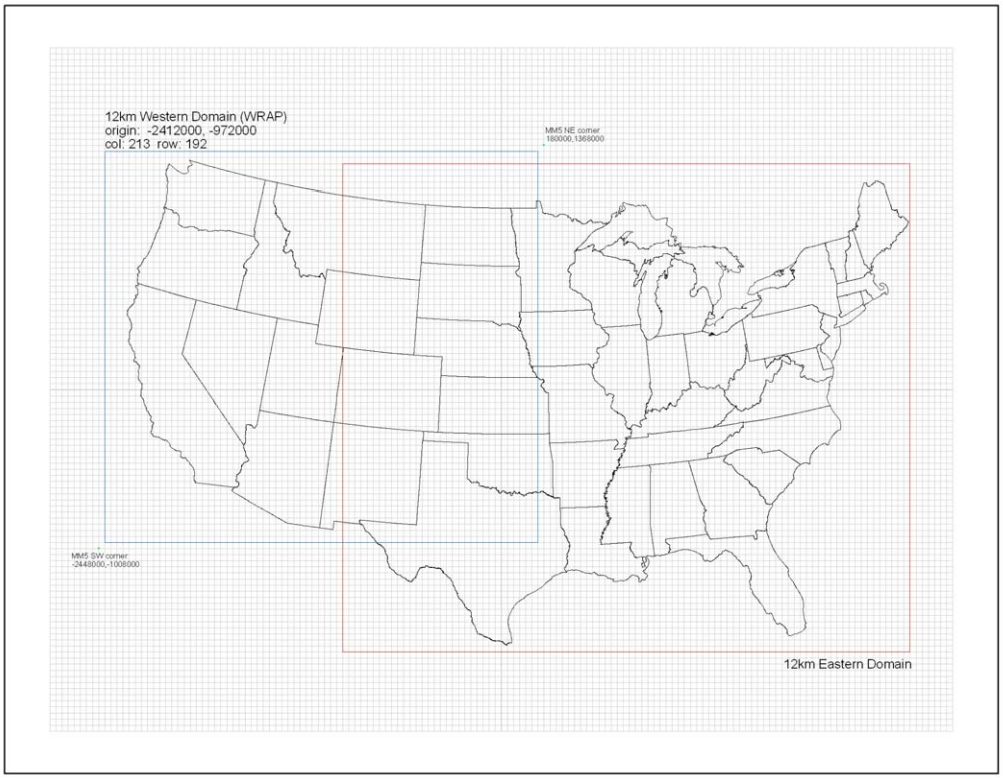

Figure 2-4. Map of the CMAQ modeling domain

2.3.3 Model Inputs

The key inputs to the CMAQ model include emissions from anthropogenic and biogenic sources, meteorological data, and initial and boundary conditions. The CMAQ meteorological input files were derived from a simulation of the Pennsylvania State University / National Center for Atmospheric Research Mesoscale Model[116] for the entire year of 2002. This model, commonly referred to as MM5, is a limited-area, nonhydrostatic, terrain-following system that solves for the full set of physical and thermodynamic equations which govern atmospheric motions. The meteorology for the national 36 km grid and the 12 km Eastern U.S. grid were developed by EPA and are described in more detail within the AQM TSD. The meteorology for the 12 km Western U.S. grid was developed by the Western Regional Air Partnership (WRAP) Regional Planning Organization. The meteorological outputs from MM5 were processed to create model-ready inputs for CMAQ using the Meteorology-Chemistry Interface Processor (MCIP) version 3.1 to derive the specific inputs to CMAQ, for example: horizontal wind components (i.e., speed and direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each grid cell in each vertical layer.[117]

The lateral boundary and initial species concentrations are provided by a three-dimensional global atmospheric chemistry model, the GEOS-CHEM model.[118] The global GEOS-CHEM model simulates atmospheric chemical and physical processes driven by assimilated meteorological observations from the NASA's Goddard Earth

Observing System (GEOS). This model was run for 2002 with a grid resolution of 2 degree x 2.5 degree (latitude-longitude) and 20 vertical layers. The predictions were used to provide one-way dynamic boundary conditions at three-hour intervals and an initial concentration field for the 36 km CMAQ simulations. The future base conditions from the 36 km coarse grid modeling were used as the initial/boundary state for all subsequent 12 km finer grid modeling.

The emissions inputs used for the 2002 base year and each of the future year base cases and control scenarios are summarized in Chapter 3 of this RIA.

2.3.4 CMAQ Evaluation

An operational model performance evaluation for $PM_{2.5}$ and its related speciated components (e.g., sulfate, nitrate, elemental carbon, organic carbon, etc.) was conducted using the 2002 data in order to estimate the ability of the CMAQ modeling system to replicate base year concentrations. In summary, model performance statistics were calculated for observed/predicted pairs of daily/monthly/seasonal/annual concentrations. Statistics were generated for the following geographic groupings: domain wide, Eastern vs. Western (divided along the 100th meridian), and each Regional Planning Organization (RPO) region.[K] The "acceptability" of model performance was judged by comparing our results to those found in recent regional $PM_{2.5}$ model applications for other, non-EPA studies.[L] Overall, the performance for the 2002 modeling platform is within the range of these other applications. A detailed summary of the 2002 CMAQ model performance evaluation is available within the AQM TSD.

2.3.5 Model Simulation Scenarios

As part of our analysis for this rulemaking the CMAQ modeling system was used to calculate 8-hour ozone concentrations, annual $PM_{2.5}$ concentrations, and visibility estimates for each of the following emissions scenarios:

2002 base year
2020 base line projection
2020 base line projection with small SI and marine SI controls
2030 base line projection
2030 base line projection with small SI and marine SI controls

[K] Regional Planning Organization regions include: Mid-Atlantic/Northeast Visibility Union (MANE-VU), Midwest Regional Planning Organization – Lake Michigan Air Directors Consortium (MWRPO-LADCO), Visibility Improvement State and Tribal Association of the Southeast (VISTAS), Central States Regional Air Partnership (CENRAP), and Western Regional Air Partnership (WRAP).

[L] These other modeling studies represent a wide range of modeling analyses which cover various models, model configurations, domains, years and/or episodes, chemical mechanisms, and aerosol modules.

It should be noted that the emission control scenarios used in the air quality and benefits modeling are slightly different than the emission control program being finalized. The differences reflect further refinements of the regulatory program since we performed the air quality modeling for this rule. Chapter 3 of this RIA describes the changes in the inputs and resulting emission inventories between the preliminary assumptions used for the air quality modeling and the final regulatory scenario. These refinements to the program would not significantly change the results summarized here or our conclusions drawn from this analysis.

We use the predictions from the model in a relative sense by combining the 2002 base-year predictions with predictions from each future-year scenario and applying these modeled ratios to ambient air quality observations to estimate annual $PM_{2.5}$ concentrations, 8-hour ozone concentrations, and visibility levels for each of the 2020 and 2030 scenarios. The ambient air quality observations are average conditions, on a site by site basis, for a period centered around the model base year (i.e., 2000-2004). After completing this process, we then calculated the effect of changes in PM, ozone and visibility air quality metrics resulting from this rulemaking on the health and welfare impact functions of the benefits analysis.

The projected annual $PM_{2.5}$ design values were calculated using the Speciated Modeled Attainment Test (SMAT) approach. The SMAT uses an Federal Reference Method FRM mass construction methodology that results in reduced nitrates (relative to the amount measured by routine speciation networks), higher mass associated with sulfates (reflecting water included in FRM measurements), and a measure of organic carbonaceous mass that is derived from the difference between measured $PM_{2.5}$ and its non-carbon components. This characterization of $PM_{2.5}$ mass also reflects crustal material and other minor constituents. The resulting characterization provides a complete mass balance. It does not have any unknown mass that is sometimes presented as the difference between measured $PM_{2.5}$ mass and the characterized chemical components derived from routine speciation measurements. However, the assumption that all mass difference is organic carbon has not been validated in many areas of the US. The SMAT methodology uses the following $PM_{2.5}$ species components: sulfates, nitrates, ammonium, organic carbon mass, elemental carbon, crustal, water, and blank mass (a fixed value of 0.5 $\mu g/m^3$). More complete details of the SMAT procedures can be found in the report "Procedures for Estimating Future $PM_{2.5}$ Values for the CAIR Final Rule by Application of the (Revised) Speciated Modeled Attainment Test (SMAT)".[119] For this latest analysis, several datasets and techniques were updated. These changes are fully described within the AQM TSD. The projected 8-hour ozone design values were calculated using the approach identified in EPA's guidance on air quality modeling attainment demonstrations.

2.3.6 Visibility Modeling Methodology

The modeling platform described in this section was also used to project changes in visibility. The estimate of visibility benefits was based on the projected improvement in annual average visibility at mandatory class I federal areas. There are 156 Federally

mandated Class I areas which, under the Regional Haze Rule, are required to achieve natural background visibility levels by 2064. These mandatory class I federal areas are mostly national parks, national monuments, and wilderness areas. There are currently 116 Interagency Monitoring of Protected Visual Environments (IMPROVE) monitoring sites (representing all 156 mandatory class I federal areas) collecting ambient $PM_{2.5}$ data at mandatory class I federal areas, but not all of these sites have complete data for 2002. For this analysis, we quantified visibility improvement at the 133 mandatory class I federal areas which have complete IMPROVE ambient data for 2002 or are represented by IMPROVE monitors with complete data.[M]

Visibility impairment is quantified in extinction units. Visibility degradation is directly proportional to decreases in light transmittal in the atmosphere. Scattering and absorption by both gases and particles decrease light transmittance. To quantify changes in visibility, our analysis computes a light-extinction coefficient (b_{ext}) and visual range. The light extinction coefficient is based on the work of Sisler, which shows the total fraction of light that is decreased per unit distance. This coefficient accounts for the scattering and absorption of light by both particles and gases and accounts for the higher extinction efficiency of fine particles compared to coarse particles. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon, and soil.[120]

Visual range is a measure of visibility that is inversely related to the extinction coefficient. Visual range can be defined as the maximum distance at which one can identify a black object against the horizon sky. Visual range (in units of kilometers) can be calculated from b_{ext} using the formula: Visual Range (km) = $3912/b_{ext}$ (b_{ext} units are inverse megameters [Mm^{-1}])
The future year visibility impairment was calculated using a methodology which applies modeling results in a relative sense similar to the Speciated Modeled Attainment Test (SMAT).

In calculating visibility impairment, the extinction coefficient is made up of individual component species (sulfate, nitrate, organics, etc). The predicted change in visibility is calculated as the percent change in the extinction coefficient for each of the PM species (on a daily average basis). The individual daily species extinction coefficients are summed to get a daily total extinction value. The daily extinction coefficients are converted to visual range and then averaged across all days. In this way, we can calculate annual average extinction and visual range at each IMPROVE site. Subtracting the annual average control case visual range from the base case visual range gives a projected improvement in visual range (in km) at each mandatory class I federal area. This serves as the visibility input for the benefits analysis (See Chapter X).

[M] There are 100 IMPROVE sites with complete data for 2002. Many of these sites collect data that is "representative" of other nearby unmonitored mandatory class I federal areas. There are a total of 133 mandatory class I federal areas that are represented by the 100 sites. The matching of sites to monitors is taken from "Guidance for Tracking Progress Under the Regional Haze Rule".

For visibility calculations, we are continuing to use the IMPROVE program species definitions and visibility formulas which are recommended in the modeling guidance.[121] Each IMPROVE site has measurements of $PM_{2.5}$ species and therefore we do not need to estimate the species fractions in the same way that we did for FRM sites (using interpolation techniques and other assumptions concerning volatilization of species).

2.4 Air Toxics

Small SI and Marine SI emissions contribute to ambient levels of air toxics known or suspected as human or animal carcinogens, or that have noncancer health effects. The population experiences an elevated risk of cancer and other noncancer health effects from exposure to air toxics.[122] These compounds include, but are not limited to, benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, polycyclic organic matter (POM), and naphthalene. These compounds, except acetaldehyde, were identified as national or regional risk drivers in the 1999 National-Scale Air Toxics Assessment (NATA) and have significant inventory contributions from mobile sources.

Table2-7 Mobile Source Inventory Contribution to 1999 Emissions of NATA Risk Drivers[a]

1999 NATA Risk Driver	Percent of Emissions Attributable to All Mobile Sources	Percent of Emissions Attributable to Non-road Sources
Benzene	68%	19%
1,3-Butadiene	58%	17%
Formaldehyde	47%	20%
Acrolein	25%	11%
Polycyclic organic matter (POM)[b]	5%	2%
Naphthalene	27%	6%
Diesel PM and Diesel exhaust organic gases	100%	62%

[a] This table is generated from data contained in the pollutant specific Microsoft Access database files found in the County-Level Emission Summaries section of the 1999 NATA webpage (http://www.epa.gov/ttn/atw/nata1999/tables.html).

[b] This POM inventory includes the 15 POM compounds: benzo[b]fluoranthene, benz[a]anthracene, indeno(1,2,3-c,d)pyrene, benzo[k]fluoranthene, chrysene, benzo[a]pyrene, dibenz(a,h)anthracene, anthracene, pyrene, benzo(g,h,i)perylene, fluoranthene, acenaphthylene, phenanthrene, fluorine, and acenaphthene.

According to NATA for 1999, mobile sources were responsible for 44 percent of outdoor toxic emissions and almost 50 percent of the cancer risk. Benzene is the largest contributor to cancer risk of all 133 pollutants quantitatively assessed in the 1999 NATA and mobile sources were responsible for 68 percent of benzene emissions in 1999. In

response, EPA has recently finalized vehicle and fuel controls that address this public health risk.[N]

People are exposed to toxics from spark-ignition engines as a result of operating these engines and from intrusion into the home of emissions that occur in residential attached garages. A study of aldehyde exposures among lawn and garden equipment operators found formaldehyde and acetaldehyde exposure concentrations, during approximately 30 to 120 minutes of engine use, that were one to two orders of magnitude greater than those measured at an upwind monitor.[123] The study also reported measurable concentrations of transition metals emitted from most test engines, in addition to high organic carbon concentrations in $PM_{2.5}$ samples. Analyses of organic material emitted from hand-held engines have detected PAHs and other compounds, suggesting that exposures to hand-held engine emissions are similar in composition to those found in motor vehicle-affected environments, such as near major roadways.[124] Numerous studies have reported elevated benzene concentrations in residential attached garages.[125,126,127] These studies indicate the potential for elevated exposures as a result of the use and storage of small spark-ignition engines.

Noncancer health effects can result from chronic,[O] subchronic,[P] or acute[Q] inhalation exposures to air toxics, and include neurological, cardiovascular, liver, kidney, and respiratory effects as well as effects on the immune and reproductive systems. According to the 1999 NATA, nearly the entire U.S. population was exposed to an average concentration of air toxics that has the potential for adverse noncancer respiratory health effects. This will continue to be the case in 2030, even though toxics concentrations will be lower. Mobile sources were responsible for 74 percent of the noncancer (respiratory) risk from outdoor air toxics in 1999. The majority of this risk was from exposure to acrolein. The confidence in the RfC for acrolein is medium and confidence in NATA estimates of population noncancer hazard from ambient exposure to this pollutant is low.[128,129]

The NATA modeling framework has a number of limitations which prevent its use as the sole basis for setting regulatory standards. These limitations and uncertainties are discussed on the 1999 NATA website.[130] Even so, this modeling framework is very

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[N] U.S. EPA (2007) Control of Hazardous Air Pollutants from Mobile Sources. 72 FR 8428; February 26, 2007.

[O] Chronic exposure is defined in the glossary of the Integrated Risk Information (IRIS) database (http://www.epa.gov/iris) as repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).

[P] Defined in the IRIS database as repeated exposure by the oral, dermal, or inhalation route for more than 30 days, up to approximately 10% of the life span in humans (more than 30 days up to approximately 90 days in typically used laboratory animal species)..

[Q] Defined in the IRIS database as exposure by the oral, dermal, or inhalation route for 24 hours or less.

useful in identifying air toxic pollutants and sources of greatest concern, setting regulatory priorities, and informing the decision making process.

Benzene: The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia) by all routes of exposure, and concludes that exposure is associated with additional health effects, including genetic changes in both humans and animals and increased proliferation of bone marrow cells in mice.[131,132,133] EPA states in its IRIS database that data indicate a causal relationship between benzene exposure and acute lymphocytic leukemia and suggest a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. The International Agency for Research on Carcinogens (IARC) has determined that benzene is a human carcinogen and the U.S. Department of Health and Human Services (DHHS) has characterized benzene as a known human carcinogen.[134,135]

A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with long-term exposure to benzene.[136,137] The most sensitive noncancer effect observed in humans, based on current data, is the depression of the absolute lymphocyte count in blood.[138,139] In addition, recent work, including studies sponsored by the Health Effects Institute (HEI), provides evidence that biochemical responses are occurring at lower levels of benzene exposure than previously known.[140,141,142,143] EPA's IRIS program has not yet evaluated these new data.

1,3-Butadiene: EPA has characterized 1,3-butadiene as carcinogenic to humans by inhalation.[144,145] The IARC has determined that 1,3-butadiene is a human carcinogen and the U.S. DHHS has characterized 1,3-butadiene as a known human carcinogen.[146,147] There are numerous studies consistently demonstrating that 1,3-butadiene is metabolized into genotoxic metabolites by experimental animals and humans. The specific mechanisms of 1,3-butadiene-induced carcinogenesis are unknown; however, the scientific evidence strongly suggests that the carcinogenic effects are mediated by genotoxic metabolites. Animal data suggest that females may be more sensitive than males for cancer effects associated with 1,3-butadiene exposure; there are insufficient data in humans from which to draw conclusions about sensitive subpopulations. 1,3-butadiene also causes a variety of reproductive and developmental effects in mice; no human data on these effects are available. The most sensitive effect was ovarian atrophy observed in a lifetime bioassay of female mice.[148]

Formaldehyde: Since 1987, EPA has classified formaldehyde as a probable human carcinogen based on evidence in humans and in rats, mice, hamsters, and monkeys.[149] EPA is currently reviewing recently published epidemiological data. For instance, research conducted by the National Cancer Institute (NCI) found an increased risk of nasopharyngeal cancer and lymphohematopoietic malignancies such as leukemia among workers exposed to formaldehyde.[150,151] NCI is currently performing an update of these studies. A recent National Institute of Occupational Safety and Health (NIOSH) study of garment workers also found increased risk of death due to leukemia among

workers exposed to formaldehyde.[152] Extended follow-up of a cohort of British chemical workers did not find evidence of an increase in nasopharyngeal or lymphohematopoietic cancers, but a continuing statistically significant excess in lung cancers was reported.[153]

In the past 15 years there has been substantial research on the inhalation dosimetry for formaldehyde in rodents and primates by the CIIT Centers for Health Research (formerly the Chemical Industry Institute of Toxicology), with a focus on use of rodent data for refinement of the quantitative cancer dose-response assessment.[154,155,156] CIIT's risk assessment of formaldehyde incorporated mechanistic and dosimetric information on formaldehyde.

Based on the developments of the last decade, in 2004, the working group of the International Agency for Research on Cancer (IARC) concluded that formaldehyde is carcinogenic to humans (Group 1), on the basis of sufficient evidence in humans and sufficient evidence in experimental animals - a higher classification than previous IARC evaluations. After reviewing the currently available epidemiological evidence, the IARC (2006) characterized the human evidence for formaldehyde carcinogenicity as "sufficient," based upon the data on nasopharyngeal cancers; the epidemiologic evidence on leukemia was characterized as "strong."[157] EPA is reviewing the recent work cited above from the NCI and NIOSH, as well as the analysis by the CIIT Centers for Health Research and other studies, as part of a reassessment of the human hazard and dose-response associated with formaldehyde.

Formaldehyde exposure also causes a range of noncancer health effects, including irritation of the eyes (burning and watering of the eyes), nose and throat. Effects from repeated exposure in humans include respiratory tract irritation, chronic bronchitis and nasal epithelial lesions such as metaplasia and loss of cilia. Animal studies suggest that formaldehyde may also cause airway inflammation – including eosinophil infiltration into the airways. There are several studies that suggest that formaldehyde may increase the risk of asthma – particularly in the young.[158,159]

Acetaldehyde: Acetaldehyde is classified in EPA's IRIS database as a probable human carcinogen, based on nasal tumors in rats, and is considered toxic by the inhalation, oral, and intravenous routes.[160] Acetaldehyde is reasonably anticipated to be a human carcinogen by the U.S. DHHS in the 11th Report on Carcinogens and is classified as possibly carcinogenic to humans (Group 2B) by the IARC.[161,162] EPA is currently conducting a reassessment of cancer risk from inhalation exposure to acetaldehyde.

The primary noncancer effects of exposure to acetaldehyde vapors include irritation of the eyes, skin, and respiratory tract.[163] In short-term (4 week) rat studies, degeneration of olfactory epithelium was observed at various concentration levels of acetaldehyde exposure.[164,165] Data from these studies were used by EPA to develop an inhalation reference concentration. Some asthmatics have been shown to be a sensitive subpopulation to decrements in functional expiratory volume (FEV1 test) and bronchoconstriction upon acetaldehyde inhalation.[166] The agency is currently conducting a reassessment of the health hazards from inhalation exposure to acetaldehyde.

Acrolein: EPA determined in 2003 that the human carcinogenic potential of acrolein could not be determined because the available data were inadequate. No information was available on the carcinogenic effects of acrolein in humans and the animal data provided inadequate evidence of carcinogenicity.[167] The IARC determined in 1995 that acrolein was not classifiable as to its carcinogenicity in humans.[168]

Acrolein is extremely acrid and irritating to humans when inhaled, with acute exposure resulting in upper respiratory tract irritation, mucus hypersecretion and congestion. Levels considerably lower than 1 ppm (2.3 mg/m^3) elicit subjective complaints of eye and nasal irritation and a decrease in the respiratory rate.[169,170] Lesions to the lungs and upper respiratory tract of rats, rabbits, and hamsters have been observed after subchronic exposure to acrolein. Based on animal data, individuals with compromised respiratory function (e.g., emphysema, asthma) are expected to be at increased risk of developing adverse responses to strong respiratory irritants such as acrolein. This was demonstrated in mice with allergic airway-disease by comparison to non-diseased mice in a study of the acute respiratory irritant effects of acrolein.[171]

EPA is currently in the process of conducting an assessment of acute exposure effects for acrolein. The intense irritancy of this carbonyl has been demonstrated during controlled tests in human subjects, who suffer intolerable eye and nasal mucosal sensory reactions within minutes of exposure.[172]

Polycyclic Organic Matter (POM): POM is generally defined as a large class of organic compounds which have multiple benzene rings and a boiling point greater than 100 degrees Celsius. Many of the compounds included in the class of compounds known as POM are classified by EPA as probable human carcinogens based on animal data. One of these compounds, naphthalene, is discussed separately below. Polycyclic aromatic hydrocarbons (PAHs) are a subset of POM that contain only hydrogen and carbon atoms. A number of PAHs are known or suspected carcinogens. Recent studies have found that maternal exposures to PAHs (a subclass of POM) in a population of pregnant women were associated with several adverse birth outcomes, including low birth weight and reduced length at birth, as well as impaired cognitive development at age three.[173,174] EPA has not yet evaluated these recent studies.

Naphthalene: Naphthalene is found in small quantities in gasoline and diesel fuels. Naphthalene emissions have been measured in larger quantities in both gasoline and diesel exhaust compared with evaporative emissions from mobile sources, indicating it is primarily a product of combustion. EPA recently released an external review draft of a reassessment of the inhalation carcinogenicity of naphthalene based on a number of recent animal carcinogenicity studies.[175] The draft reassessment recently completed external peer review.[176] Based on external peer review comments received to date, additional analyses are being undertaken. This external review draft does not represent official agency opinion and was released solely for the purposes of external peer review and public comment. Once EPA evaluates public and peer reviewer comments, the document will be revised. The National Toxicology Program listed naphthalene as

"reasonably anticipated to be a human carcinogen" in 2004 on the basis of bioassays reporting clear evidence of carcinogenicity in rats and some evidence of carcinogenicity in mice.[177] California EPA has released a new risk assessment for naphthalene, and the IARC has reevaluated naphthalene and re-classified it as Group 2B: possibly carcinogenic to humans.[178] Naphthalene also causes a number of chronic non-cancer effects in animals, including abnormal cell changes and growth in respiratory and nasal tissues.[179]

The small SI and marine SI standards will reduce air toxics emitted from these engines, vessels and equipment, thereby helping to mitigate some of the adverse health effects associated with their operation. The assumption that toxic reductions track reductions in HC are supported by results from numerous test programs, including recent testing on small nonroad gasoline engines with and without controls.[180]

2.5 Carbon Monoxide

Unlike many gases, CO is odorless, colorless, tasteless, and nonirritating. Carbon monoxide results from incomplete combustion of fuel and is emitted directly from vehicle tailpipes. Incomplete combustion is most likely to occur at low air-to-fuel ratios in the engine. These conditions are common during vehicle starting when air supply is restricted ("choked"), when vehicles are not tuned properly, and at high altitude, where "thin" air effectively reduces the amount of oxygen available for combustion (except in engines that are designed or adjusted to compensate for altitude). High concentrations of CO generally occur in areas with elevated mobile-source emissions. Carbon monoxide emissions increase dramatically in cold weather. This is because engines need more fuel to start at cold temperatures and because some emission control devices (such as oxygen sensors and catalytic converters) operate less efficiently when they are cold. Also, nighttime inversion conditions are more frequent in the colder months of the year. This is due to the enhanced stability in the atmospheric boundary layer, which inhibits vertical mixing of emissions from the surface.

2.5.1 Health Effects of CO Pollution

We are relying on the data and conclusions in the EPA Air Quality Criteria Document for CO (CO Criteria Document), which was published in 2000, regarding the health effects associated with CO exposure.[R181] Carbon monoxide enters the bloodstream through the lungs and forms carboxyhemoglobin (COHb), a compound that inhibits the blood's capacity to carry oxygen to organs and tissues.[182,183] Carbon monoxide has long been known to have substantial adverse effects on human health, including toxic effects on blood and tissues, and effects on organ functions. Although there are effective compensatory increases in blood flow to the brain, at some concentrations of COHb, somewhere above 20 percent, these compensations fail to maintain sufficient oxygen delivery, and metabolism declines.[184] The subsequent hypoxia in brain tissue then

[R] The NAAQS review process is underway for CO and the CO Integrated Science Assessment is scheduled to be completed in 2010.

produces behavioral effects, including decrements in continuous performance and reaction time.[185]

Carbon monoxide has been linked to increased risk for people with heart disease, reduced visual perception, cognitive functions and aerobic capacity, and possible fetal effects.[186] Persons with heart disease are especially sensitive to carbon monoxide poisoning and may experience chest pain if they breathe the gas while exercising.[187] Infants, elderly persons, and individuals with respiratory diseases are also particularly sensitive. Carbon monoxide can affect healthy individuals, impairing exercise capacity, visual perception, manual dexterity, learning functions, and ability to perform complex tasks.[188]

Several epidemiological studies have shown a link between CO and premature morbidity (including angina, congestive heart failure, and other cardiovascular diseases). Several studies in the United States and Canada have also reported an association between ambient CO exposures and frequency of cardiovascular hospital admissions, especially for congestive heart failure (CHF). An association between ambient CO exposure and mortality has also been reported in epidemiological studies, though not as consistently or specifically as with CHF admissions. EPA reviewed these studies as part of the CO Criteria Document review process and noted the possibility that the average ambient CO levels used as exposure indices in the epidemiology studies may be surrogates for ambient air mixes impacted by combustion sources and/or other constituent toxic components of such mixes. More research will be needed to better clarify CO's role.[189]

As noted above, CO has been linked to numerous health effects. In addition to health effects from chronic exposure to ambient CO levels, acute exposures to higher levels are also a problem. Acute exposures to CO are discussed further in Section 2.6.

2.5.2 Attainment and Maintenance of the CO NAAQS

On July 3, 1995 EPA made a finding that small land-based spark-ignition engines cause or contribute to CO nonattainment (60 FR 34581, July 3, 1995). Marine spark-ignition engines, which have relatively high per engine CO emissions, can also be a source of CO emissions in CO nonattainment areas. In the preamble for this proposed rule EPA makes a finding that recreational marine engines and vessels cause or contribute to CO nonattainment and we provide information showing CO emissions from spark-ignition marine engines and vessels in the CO nonattainment areas in 2005. Spark-ignition marine engines and vessels contribute to CO nonattainment in more than one of the CO nonattainment areas.

A nonattainment area is defined in the Clean Air Act (CAA) as an area that is violating an ambient standard or is contributing to a nearby area that is violating the standard. EPA has designated nonattainment areas for the CO NAAQS by calculating air quality design values and considering other factors.[S]

[S] The full details involved in calculating a CO design value are given in 40 CFR Part 50.8.

There are two CO NAAQS. The 8-hour average CO NAAQS is 9 ppm, not to be exceeded more than once per year, and the 1-hour average CO NAAQS is 35 ppm, not to be exceeded more than once per year. As of March 12, 2008, there are approximately 850 thousand people living in 4 areas (which include 5 counties) that are designated as nonattainment for CO, see Table 2-8. The emission reductions in this rule will help areas to attain and maintain the CO NAAQS.

Table 2-8: Classified Carbon Monoxide Nonattainment Areas as of March 2008[a]

Area	Classification	Population (1000s)
Las Vegas, NV	serious	479
El Paso, TX	moderate <= 12.7 ppm	62
Reno, NV	moderate <= 12.7 ppm	179
Total		719.5

[a] This table does not include Salem, OR which is an unclassified CO nonattainment area.

In addition to the CO nonattainment areas, there are areas that have not been designated as nonattainment where air quality monitoring may indicate a need for CO control. For example, areas like Birmingham, AL and Calexico, CA have not been designated as nonattainment although monitors in these areas have recorded multiple exceedances since 1995.[190]

There are also almost 69 million people living in CO maintenance areas, see Table 2-9.[T] Carbon monoxide maintenance areas may remain at risk for high CO episodes especially in geographic areas with unusually challenging meteorological and topographical conditions and in areas with high population growth and increasing vehicle miles traveled.

Table 2-9: Carbon Monoxide Maintenance Areas as of March 2008

	Number of Areas	Number of Counties	Population (1000s)
Serious	6	15	20,496,077
Moderate > 12.7ppm	4	19	17,575,606
Moderate <= 12.7ppm	30	62	23,371,653
Unclassified	33	38	7,480,907
Total	73	127	68,924,243

A 2003 NAS report found that in geographical areas that have achieved attainment of the NAAQS, it might still be possible for ambient concentrations of CO to sporadically exceed the standard under unfavorable conditions such as strong winter

[T] The CO nonattainment and maintenance areas are listed in Appendix 2E to this RIA.

inversions. Areas like Alaska are prone to winter inversions due to their topographic and meteorological conditions. The report further suggests that additional reductions in CO are prudent to further reduce the risk of violations in regions with problematic topography and temporal variability in meteorology.[191] The reductions in CO emissions from this rule will assist areas in maintaining the CO standard.

As discussed in the preamble, Small SI engines and equipment and Marine SI engines and vessels do contribute to CO nonattainment. The CO emission benefits from this rule will help states in their strategy to attain the CO NAAQS. Maintenance of the CO NAAQS is also challenging and many areas will be able to use the emissions reductions from this rule to assist in maintaining the CO NAAQS into the future.

2.6 Acute Exposure to Air Pollutants

Emissions from Small SI engines and equipment and Marine SI engines and vessels contribute to ambient concentrations of ozone, CO, air toxics and PM and acute exposures to air toxics, CO and PM. The standards being finalized in this action can help reduce acute exposures to emissions from Marine SI engines and vessels and Small SI engines and equipment.

2.6.1 Exposure to CO from Marine SI Engines and Vessels

In recent years, a substantial number of CO poisonings and deaths have occurred on and around recreational boats across the nation. The actual number of deaths attributable to CO poisoning while boating is difficult to estimate because CO-related deaths in the water may be labeled as drowning. An interagency team consisting of the National Park Service, the U.S. Department of Interior, and the National Institute for Occupational Safety and Health maintains a record of published CO-related fatal and nonfatal poisonings.[192] Between 1984 and 2004, 113 CO-related deaths and 458 non-fatal CO poisonings have been identified based on hospital records, press accounts, and other information. Deaths have been attributed to exhaust from both onboard generators and propulsion engines. Houseboats, cabin cruisers, and ski boats are the most common types of boats associated with CO poisoning cases. These incidents have prompted other federal agencies, including the United States Coast Guard and National Park Service, to issue advisory statements and other interventions to boaters to avoid activities that could lead to excessive CO exposure.[193]

CO concentrations can be extremely elevated within several meters of the exhaust port. Engineers and industrial hygienists from CDC/NIOSH and other state and federal agencies have conducted field studies of CO concentrations on and around houseboats. In one study of houseboat concentrations, CO concentrations immediately at the point of generator exhaust discharge on one houseboat averaged 0.5% (5,000 ppm), and ranged from 0.0% to 1.28% (12,800 ppm).[194] With both propulsion and generators running, time-averaged concentrations on the swim deck were 0.2 - 169 ppm at different locations on one boat's swim platform, 17-570 ppm on another's, and 0-108 on another. Other studies also show the potential for high concentrations with extreme peaks in CO

concentrations in locations where boaters and swimmers can be exposed during typical boating activities, such as standing on a swim deck or swimming near a boat.

2.6.2 Exposure to CO and PM from Small SI Engines and Equipment

A large segment of the population uses small, gasoline-powered SI lawn and garden equipment on a regular basis. Emissions from many of the Small SI engines powering this equipment may lead to elevated air pollution exposures for a number of gaseous and particulate compounds, especially for individuals such as landscapers, whose occupations require the daily use of these engines and equipment.

Emission studies with lawn and garden equipment suggest a potential for high exposures during the Small SI engine operation.[195,196] Studies investigating air pollutant exposures during small engine use did report elevated personal exposure measurements related to lawn and garden equipment use.[197,198] Bunger et al. reported elevated CO personal measurements related to chainsaw use, with short-term concentrations exceeding 400 ppm for certain cutting activities. This study evaluated personal exposures during the use of uncontrolled chainsaws. Baldauf at al. evaluated the use of lawnmowers, chainsaws and string trimmers meeting US EPA Phase 2 standards. In this study, short-term exposures during lawnmower and chainsaw use exceeded 120 ppm of CO, while string trimmer use resulted in some short-term exposures approaching 100 ppm of CO. This study also indicated that short-term $PM_{2.5}$ exposures could exceed 100 $\mu g/m^3$. Pollutant exposures were highly dependent on the operator's orientation to the engine and wind direction, as well as the activities being conducted.

These studies indicate that emissions from some lawn and garden equipment meeting EPA's current Phase 2 standards may contribute to elevated exposures to certain pollutants. The potential for elevated exposure to CO and $PM_{2.5}$ for operators of Small SI engines and equipment will be reduced by this rule.

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[1] U.S. EPA. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final). U.S. EPA, Washington, DC, EPA/600/R-05/004aF-cF, 2006. **Error! Main Document Only.**This document is contained in Docket Identification EPA-HQ-OAR-2004-0008-0455 to 0457.

[2] U.S. EPA. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final). U.S. EPA, Washington, DC, EPA/600/R-05/004aF-cF, 2006. **Error! Main Document Only.**This document is contained in Docket Identification EPA-HQ-OAR-2004-0008-0455 to 0457.

[3] U.S. EPA, Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper, Washington, DC, EPA-452/R-07-003, January

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[11] U.S. EPA. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Final). U.S. EPA, Washington, DC, EPA/600/R-05/004aF-cF, 2006. This document is contained in Docket Identification EPA-HQ-OAR-2004-0008-0455 to 0457.

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